A PROSPECTIVE STUDY OF COGNITIVE ASSESSMENT IN PSYCHIATRIC PATIENTS BEFORE AND AFTER ELECTROCONVULSIVE THERAPY

Dissertation submitted to

THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY

In partial fulfillment for the requirements for

DOCTOR OF MEDICINE

(BRANCH – XVIII) PSYCHIATRY

EXAMINATIONS – APRIL 2016

DEPARTMENT OF PSYCHIATRY,

TIRUNELVELI MEDICAL COLLEGE AND HOSPITAL

TIRUNELVELI – 627 011

A PROSPECTIVE STUDY OF COGNITIVE ASSESSMENT IN PSYCHIATRIC PATIENTS BEFORE AND AFTER ELECTROCONVULSIVE THERAPY

Dissertation submitted to

THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY

In partial fulfillment for the requirements for

DOCTOR OF MEDICINE

(BRANCH – XVIII) PSYCHIATRY

EXAMINATIONS – APRIL 2016

DEPARTMENT OF PSYCHIATRY,

TIRUNELVELI MEDICAL COLLEGE AND HOSPITAL

TIRUNELVELI – 627 011

CERTIFICATE

This is to certify that this dissertation titled "**A PROSPECTIVE STUDY OF COGNITIVE ASSESSMENT IN PSYCHIATRIC PATIENTS BEFORE AND AFTER ELECTROCONVULSIVE THERAPY"** submitted by **Dr.M.Asif Abrar,** appearing for **M.D (Psychiatry)** degree examination in April 2016 is a original bonafide record of work done from January 2014 to June 2015 by him under my guidance and supervision in partial fulfillment of requirements of the TamilNadu Dr.M.G.R.Medical University, Chennai. I forward this to the TamilNadu Dr.M.G.R.Medical University, Chennai, TamilNadu, India.

Dr.M.B.Abdul Rahman,M.D.,
Assistant Professor,
Department of Psychiatry
Tirunelveli Medical College,
Tirunelveli

Dr.G.Ramanujam, M.D.,
Associate Professor & HOD,
Department of Psychiatry
Tirunelveli Medical College,
Tirunelveli

Dr. SITHY ATHIYA MUNAVARAH

The Dean,
Tirunelveli Medical College,
Tirunelveli

DECLARATION

I, Dr.M.Asif Abrar, solemnly declare that this dissertation "**A PROSPECTIVE STUDY OF COGNITIVE ASSESSMENT IN PSYCHIATRIC PATIENTS BEFORE AND AFTER ELECTROCONVULSIVE THERAPY"** was done by me at the Department of Psychiatry, Tirunelveli Medical College, Tirunelveli under the guidance and supervision of the Professor of Psychiatry, Tirunelveli Medical College, Tirunelveli between January 2014 and June 2015.

The dissertation is submitted to the TamilNadu Dr.M.G.R Medical University, Chennai-32 in partial fulfillment of the University requirements for the award of the degree of MD., Psychiatry.

Place: Tirunelveli

Date: (Dr.M.Asif Abrar)

ACKNOWLEDGEMENT

I owe my thanks to THE DEAN, Tirunelveli Medical College, Tirunelveli for permitting me to utilize the facilities and clinical materials for conducting this study.

I am extremely grateful to Associate Professor of Psychiatry, Dr.G.Ramanujam, Tirunelveli Medical College, Tirunelveli, for his constant encouragement and guidance throughout the study and for his periodic reviews.

I am indebted to Dr.M.B.Abdul Rahman, Assistant Professor for his support, guidance and help without which it would have been difficult to carry out this study.

I am extremely thankful to Dr.A.Godson and Dr.S.Jeeva Creedom Victory for helping me with their time and advice during the study.

I wish to thank the paramedical and non medical staff of the Department of Psychiatry for their cooperation which helped me in this study.

I thank all the patients who consented to participate in this study without which this study would not have been possible.

The blessings of God and support of my family needs special mention.

TIRUNELVELI MEDICAL COLLEGE

INSTITUTIONAL RESEARCH ETHICS COMMITTEE

TIRUNELVELI, STATE OF TAMILNADU, SOUTH INDIA PIN 627011
91-462-2572733-EXT; 91-462-2572944; 91-462-2579785; 91-462-2572611-16
online@tvmc.ac.in, tirec@tvmc.ac.in; www.tvmc.ac.in

CERTIFICATE OF REGISTRATION & APPROVAL OF THE TIREC REF NO: 474/PSYCH /2013/24

PROTOCOL TITLE: A prospective study of cognitive assessment in psychiatric patients before and after electro convulsive therapy

NAME OF PRINCIPAL INVESTIGATOR: Dr. M.Asif Abrar
DESIGNATION OF PRINCIPAL INVESTIGATOR: Resident in Psychiatry
DEPARTMENT & INSTITUTION: Department of Psychiatry, Tirunelveli Medical College

Dear Dr. M.Asif Abrar, *The Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during the IEC meeting held on 28.12.13.*

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1.	TIREC Application Form	☑
2.	Study Protocol	☑
3.	Department Research Committee Approval	☑
4.	Patient Information Document and Consent Form in English and Vernacular Language	☑
5.	Investigator's Brochure	☐
6.	Proposed Methods for Patient Accrual Proposed	☑
7.	Curriculum Vitae of the Principal Investigator	☑
8.	Insurance /Compensation Policy	☑
9.	Investigator's Agreement with Sponsor	☐
10.	Investigator's Undertaking	☑
11.	DCGI/DGFT approval	☐
12.	Clinical Trial Agreement (CTA)	☐
13.	Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA)	☑
14.	Clinical Trials Registry-India (CTRI) Registration	☐

THE PROTOCOL IS APPROVED IN ITS PRESENTED FORM ON THE FOLLOWING CONDITIONS

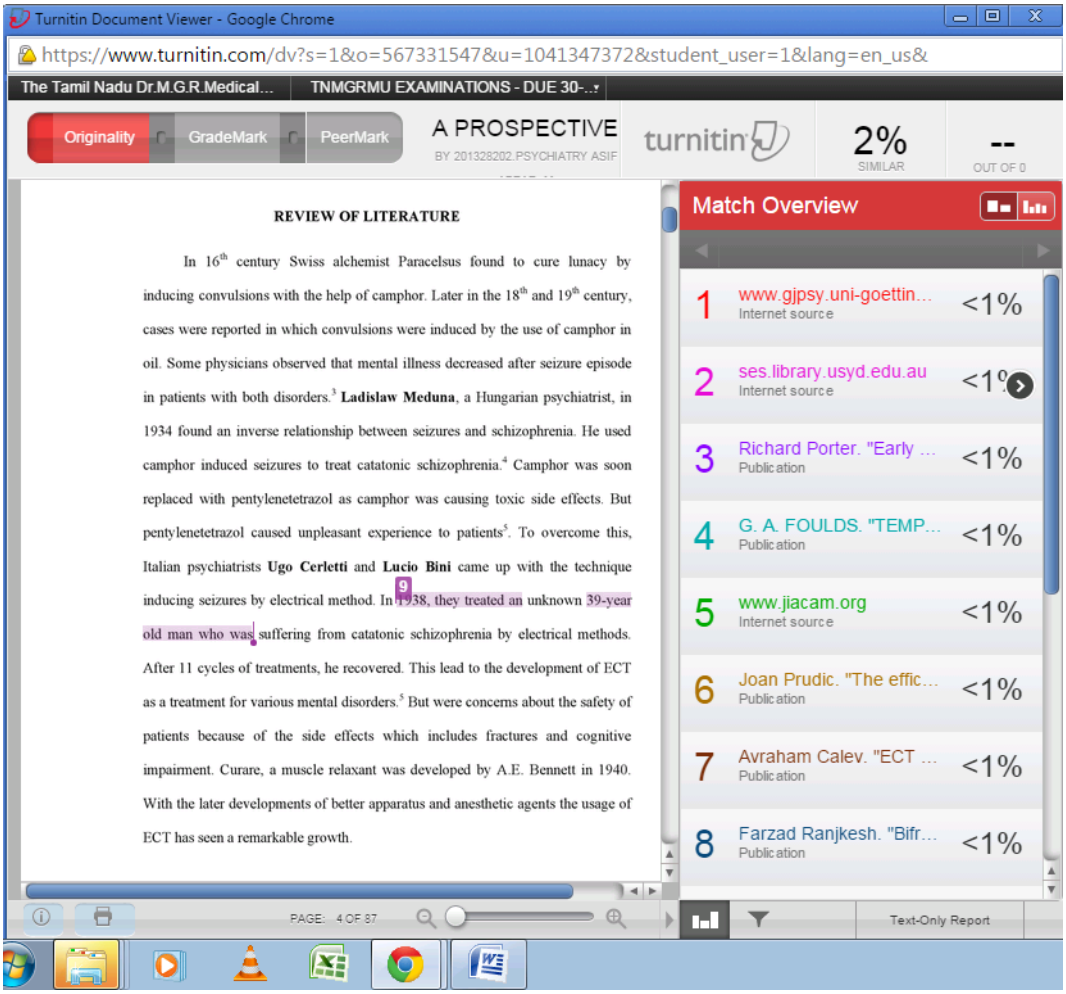
1. The approval is valid for a period of 2 year/s or duration of project whichever is later
2. The date of commencement of study should be informed
3. A written request should be submitted 3weeks before for renewal / extension of the validity
4. An annual status report should be submitted.
5. The TIREC will monitor the study
6. At the time of PI's retirement/leaving the institute, the study responsibility should be transferred to a person cleared by HOD
7. The PI should report to TIREC within 7 days of the occurrence of the SAE. If the SAE is Death, the Bioethics Cell should receive the SAE reporting form within 24 hours of the occurrence.
8. In the events of any protocol amendments, TIREC must be informed and the amendments should be highlighted in clear terms as follows:
   a. The exact alteration/amendment should be specified and indicated where the amendment occurred in the original project. (Page no. Clause no. etc.)
   b. The PI must comment how proposed amendment will affect the ongoing trial. Alteration in the budgetary status, staff requirement should be clearly indicated and the revised budget form should be submitted.
   c. If the amendments require a change in the consent form, the copy of revised Consent Form should be submitted to Ethics Committee for approval. If the amendment demands a re-look at the toxicity or side effects to patients, the same should be documented.
   d. If there are any amendments in the trial design, these must be incorporated in the protocol, and other study documents. These revised documents should be submitted for approval of the IEC, only then can they be implemented.
   e. Approval for amendment changes must be obtained prior to implementation of changes.
   f. The amendment is unlikely to be approved by the IEC unless all the above information is provided.
   g. Any deviation/violation/waiver in the protocol must be informed

STANDS APPROVED UNDER SEAL

Dr.K.Shantaraman MD
Registrar, TIREC

TIRUNELVELI MEDICAL COLLEGE

Dr.V.Ramasubramanian MD DM
Member Secretary, TIREC


Turnitin Document Viewer - Google Chrome
https://www.turnitin.com/dv?s=1&o=567331547&u=1041347372&student_user=1&lang=en_us&
The Tamil Nadu Dr.M.G.R.Medical...
TNMGRMU EXAMINATIONS - DUE 30-...
Originality
GradeMark
PeerMark
A PROSPECTIVE
BY 201328202.PSYCHIATRY ASIF
turnitin
2%
SIMILAR
--
OUT OF 0
REVIEW OF LITERATURE
In 16th century Swiss alchemist Paracelsus found to cure lunacy by inducing convulsions with the help of camphor. Later in the 18th and 19th century, cases were reported in which convulsions were induced by the use of camphor in oil. Some physicians observed that mental illness decreased after seizure episode in patients with both disorders.[3] Ladislaw Meduna, a Hungarian psychiatrist, in 1934 found an inverse relationship between seizures and schizophrenia. He used camphor induced seizures to treat catatonic schizophrenia.[4] Camphor was soon replaced with pentylenetetrazol as camphor was causing toxic side effects. But pentylenetetrazol caused unpleasant experience to patients[5]. To overcome this, Italian psychiatrists Ugo Cerletti and Lucio Bini came up with the technique inducing seizures by electrical method. In 1938, they treated an unknown 39-year old man who was suffering from catatonic schizophrenia by electrical methods. After 11 cycles of treatments, he recovered. This lead to the development of ECT as a treatment for various mental disorders.[5] But were concerns about the safety of patients because of the side effects which includes fractures and cognitive impairment. Curare, a muscle relaxant was developed by A.E. Bennett in 1940. With the later developments of better apparatus and anesthetic agents the usage of ECT has seen a remarkable growth.
PAGE: 4 OF 87
Match Overview
1 www.gjpsy.uni-goettin... Internet source <1%
2 ses.library.usyd.edu.au Internet source <1%
3 Richard Porter. "Early ... Publication <1%
4 G. A. FOULDS. "TEMP... Publication <1%
5 www.jiacam.org Internet source <1%
6 Joan Prudic. "The effic... Publication <1%
7 Avraham Calev. "ECT ... Publication <1%
8 Farzad Ranjkesh. "Bifr... Publication <1%
Text-Only Report

INDEX

INTRODUCTION

Electro Convulsive Therapy (ECT) is a technique involving the application of electric current with the help of electrodes that are placed in the cranial vault under anesthesia. Convulsions are induced by delivering electrical charges through the electrodes. It has been used as a therapeutic procedure for the treatment of psychiatric illness for past sixty years. ECT is reported to be beneficial for pharmacological resistant mental illnesses[1]. ECT has shown very good results in patients with catatonia, major depressive disorder, schizophrenia and mania. It is used when there is poor drug compliance or in emergency situations such as aggression, suicidality, poor intake etc,. Many physiological changes occur after ECT, one of which is diminished cognition. Effects of ECT on cognition are an area of intense debate. Some researchers claim that there will be diminished cognition particularly memory impairment after ECT while others are of the view that it will improve since mental illness declines after ECT. Effects of ECT on cognition depend upon several factors like frequency of treatment, previous cognitive functions, technique with which ECT is administered, dose of anesthetic medications. Literature and studies reports of disagreement on the relative importance of these major issues[1,2]. Effects on cognitive functions seems to be a major barrier for ECT administration and its negative views although many research are of view that these effects are mild and short lived. Wave forms, electrode placement, intensity of stimulus and pulse width are some factors which play a significant

role on cognition after ECT. The current study aims to estimate the cognitive functions in patients before and after ECT.

ECT has been constantly under criticism in both professional and public views because of the presumed ideas about the effects of ECT on cognitive functions. Most of the studies are of the opinion that these effects are mild and short lived but the opposite view regarding these effects plays a major barrier in the broad application of ECT practice. The wave forms in which stimulus is given, intensity of stimulus applied and pulse width are all known to have an impact on the cognitive functions. ECT given in brief pulse wave form has less such morbidity. A variety of technical factors like the type of electrode placement and many such factors has an impact on cognitive functions[1,2]. Even the exact mechanism of action of ECT is still inconclusive and many theories have been postulated regarding the mechanism of action.

The current study aims to assess the short term effect of ECT on cognitive functions in patients subjected to ECT. This study also compares the cognitive functions before and after administration of ECT.

RATIONALE OF THE STUDY

There is need to study the cognitive effects of ECT, because ECT is usually given as per protocol in a clinical setting. The pattern, method of placement and also intensity of stimulus influences the cognitive outcomes in the post ECT period. The study of the type and also the extent of cognitive functions before and after ECT administration will help us to evaluate the

setting at which it should be clinically given and also help us to explore the mechanism of its action still further. Finally it helps us in planning for future treatments taking the cognitive functions into account and to properly assess the risk benefit ratio before administration of ECT.

REVIEW OF LITERATURE

In 16th century Swiss alchemist Paracelsus found to cure lunacy by inducing convulsions with the help of camphor. Later in the 18th and 19th century, cases were reported in which convulsions were induced by the use of camphor in oil. Some physicians observed that mental illness decreased after seizure episode in patients with both disorders.[3] **Ladislaw Meduna**, a Hungarian psychiatrist, in 1934 found an inverse relationship between seizures and schizophrenia. He used camphor induced seizures to treat catatonic schizophrenia.[4] Camphor was soon replaced with pentylenetetrazol as camphor was causing toxic side effects. But pentylenetetrazol caused unpleasant experience to patients[5]. To overcome this, Italian psychiatrists **Ugo Cerletti** and **Lucio Bini** came up with the technique inducing seizures by electrical method. In 1938, they treated an unknown 39-year old man who was suffering from catatonic schizophrenia by electrical methods. After 11 cycles of treatments, he recovered. This lead to the development of ECT as a treatment for various mental disorders.[5] But were concerns about the safety of patients because of the side effects which includes fractures and cognitive impairment. Curare, a muscle relaxant was developed by A.E. Bennett in 1940. With the later developments of better apparatus and anesthetic agents the usage of ECT has seen a remarkable growth.

The **National Institute for Health and Care Excellence (NICE)** recommends the use of ECT for the following cases:

- For treating severe depression
- For treating catatonia
- For treating severe mania

ECT should be used as a last resort when other treatment options have failed or in the case of life threatening issues. The decision to apply ECT should involve carefully weighing the benefits and analyzing the risks for the patient. The major risks that should be considered are the usage of anesthesia and in case of other illnesses. Care should be taken when it is applied for pregnant women and elderly people as the risk involved is higher.[6]

Nowadays ECT is prescribed for patients having major depressive disorder with suicidal tendencies, depression with psychosis, catatonia, mania, schizophrenia, post-traumatic stress disorder, Parkinson's disease, delirium, tardive dyskinesia, neuroleptic malignant syndrome, intractable seizure disorder and obsessive compulsive disorder.[7]

MECHANISM OF ACTION (MOA)

Though ECT has been in existence for more than 70 years, the mechanism of action is inconclusive. Several theories have been postulated regarding its mechanism. Some of which are discussed below

- **Psychological theories**

Psychological theories include Psychoanalytic and Non-Psychoanalytic theories. According to the psychoanalytic theories the mechanism involves regression, fear and punishment theories. These theories became obsolete when muscle relaxant and anesthesia came into use for ECT as studied by **Miller E, 1967.**[8]

According to the non-psychoanalytic theories the mechanism involves brain damage and amnesia. **Summerskill et al., 1952**[9] suggested that brain damage theory was framed due to the similarities observed between the Rorschach's test results of patients after ECT and diffuses brain damage. However no change in the structure of brain was found post-ECT in 2-3 days in the study by **Coffey et al., 1989**[10]. Also no MRI changes were found for a period of 1 week as reported by **Pande et al., 1990**[11]. According to **Devanand et al., 1995**[12] human autopsy results showed no structural brain damage. Amnesia of pre-treatment experiences was common in post-ECT patients. Amnesia theory was nullified due to the lack of relation between therapeutic efficacy and amnesia in latest techniques of unilateral ECT as reported by **Lawson et al., 1990**[13].

- **Neurophysiological theories**

The MOA of ECT can be explained by theories dealing with the physiological changes caused by it, which are Sleep, Anticonvulsant and Neurogenesis theory.

EEG changes such as frequency reduction and increased amplitude of delta waves were noted after ECT. This is similar to that of normal sleep pattern. **Charlton, 1999**[14] reported that the seizure caused by ECT induces natural and deep restorative sleep. But **Weiner et al., 1984**[15] suggested that the relation of therapeutic response with EEG slowing is inconsistent. Certain studies also suggest sleep deprivation as a good antidepressant**. Eriksson et al., 1998**[16] **and Gloud et al., 1998**[17] reported that the Hippocampus is where neurogenesis occurs in humans. Studies by **Shah et al., 1998**[18] **and Sheline et al., 1999**[19] have shown decreased hippocampal volume after major depression. Depression also causes degeneration and decreased proliferation. ECT might reverse this by stimulation of neuronal proliferation.

Anticonvulsant theories are formulated using the results from ECT/Seizure disorders. Threshold for seizures increases after ECT as reported by **Kalinowsky & Kennedy, 1943**[20] which is attributed to alteration in opioid and GABA transmission by **Issac et al., 1986**.[21] GABAergic transmissions is increased post-ECT which in turn contributes to the threshold increase. Another conclusion is that ECS releases opioid like endogenous anticonvulsant substance by **Holaday et al., 1986**.[22] There was decrease in the duration of seizure after ECT by **Sackeim et al., 1986 & 1987c**.[23,24] This reduction has no association with the therapeutic response of ECT. Inhibitory process begins during the ictus and manifests to the immediate postictal period. Superior therapeutic effects have been

observed when there is high amplitude slow wave in the delta activity by **Nobler et al., 1993**.[25]The inhibitory process is linked with efficacy thus supporting the anticonvulsant theory. The Cerebral Blood Flow (CBF) and Cerebral Metabolic Rate (CMR) are reduced in anterior frontal regions and in immediate postictal period which gives very good clinical outcome by **Volkow et al., 1988**.[26]The study **by Bonne et al., in 1996**[27] opposes this fact. There is a relation between seizure threshold and CBF which supports the anticonvulsant theory. There is a substantial increase of slow waves in the inter-ictal period after application of ECT by **Sackeim et al., 1996**.[28] Various findings show that the reduction in the functional activity of a particular brain region might be the reason for the therapeutic response of ECT by **Abrams et al., 1992**.[29] The EEG coherence of anterior inter-hemispheres was found to be decreased in patients with depression which was responsible for better response of ECT. The link between such changes and the reduced response to ECT and medication was studied by **Leuchter et al., 1997**.[30]This fact gives a direct association of anticonvulsant and antidepressant.

Neurochemical theories

- **Serotonin (5HT)**

The levels of 5HT are not affected by ECT, but serotonin is found to be released as a response to therapy while using antidepressants. The variations in levels of $5HT_{1a}$ and $5HT_{2a}$ are shown below:

Receptors	ECT	Antidepressant drugs
$5HT_{1a}$	No response	Decreases
$5HT_{2a}$	Increases	Decreases

So the results are similar for ECT but are in opposition with that of antidepressant drugs.

- **Norepinephrine (NE)**

Norepinephrine levels were not altered by **Slade 1980**.[31] In beta receptor varying results have been found, beta receptors increased responsiveness on lymphocyte after ECT by **Mann et al., 1990**.[32] Post synaptic beta receptor down regulation and no change after ECT in some studies done by **Cooper et al., 1985**.[33]

Human studies revealed number of alpha-2 platelet receptor was decreased after ECT by **Smith et al., 1983.**[34]

Alpha 1 receptor number is increased in hippocampus after ECT was reported by **Vetulani et al., 1983**.[35]

- **Dopamine**

Dopamine in striatum and dopamine receptors increases after ECT. Functional polymorphism occurs in dopamine D2 receptor. This receptor is responsible for the regulation of postsynaptic effects and COMT gene

which in turn metabolizes dopamine and noradrenaline. This is linked with varying response to ECT.

- **GABA(Gamma-aminobutyric acid)**

On comparing the GABA levels of ECT responders with non-responders, it was found that the mean level of GABA was higher for ECT responders by **Devanand et al., 1995**.[36]The other factors that should be considered are the increase in glutamic acid decarboxylase, enhanced GABAergic function, augmentation of GABA receptor and increase in the Thyrotropin-Releasing Harmone (TRH).

- **Glutamate**

Long term ECT is found to cause an increase in Glutamate receptor expression and this might contribute to the better induction of brain derived neurotrophic factor (BDNF) by **Naylor et al., 1996**.[37]The adverse effects of ECT may be accounted to the activation of glutamate receptors (hippocampus) during the seizure.

- **Melatonin**

The day night ratios of melatonin levels were higher in Pre-ECT patients when compared with Post-ECT patients by **Krahn et al., 2000**.[38]Thus the findings are inconclusive.

- **Endocrine effects**

ECT produces Diencephalic stimulation as a result of which neuropeptide dysregulation is corrected by **Abrams & Taylor., 1976**.[39] ECT also increases blood drain barrier permeability which increases neuropeptide distribution through CNS. Prolactin release is higher in bilateral ECT when compared with unilateral ECT. Also it is higher in high dose stimulation when compared with low dose stimulation by **Deakin et al., 1983**.[40]There is a direct correlation between prolactin surge induced by ECT and the clinical improvement in depression **Lisanby et al., 1998**.[41]

- **Neuropeptides**

After the application of ECT, there is a considerable increase in the values of Neuropeptide Y (NPY), endothelin and somatostatin by **Mathe et al., 1999**.[42]The maximum amount of variation is seen in the hippocampus.

- **Intracellular mechanisms**

BDNF expression was regulated by ECT which is seen in dentate gyrus of hippocampus by **Lindefors et al., 1995**.[43] It is mediated by glutamate receptor activation and its expression is under negative control of GABAergic receptor activation as reported by **Metsis et al., 1993**.[44]

Other possible mechanisms include alteration of coupling of G proteins to corresponding receptors, Phospolipase and adenylyl cyclase activity and neuronal calcium entry in second messenger system, Effects of decreased

cholinergic transmission after ECT, Neurogenesis promotion and apoptosis suppression, excitatory amino acid release after ECT.

MECHANISM OF COGNITIVE AND AMNESIC EFFECTS OF ECT

Devanand et al., 1994 [45] have shown that ECT does not cause any structural brain damage. MRI studies have confirmed such findings in humans by **Coffey et al., 1991**.[46] Mechanism of post ECT amnesia is explained by excitotoxicity, glutaminergic, cholinergic, hypertensive surge, glucocorticoid, and also cyclooxygenase and other related mechanisms**.**[47]

Hippocampus plays a key role in proper retrieval and memory functioning.[48]

It is seen that the hippocampal neurons are more prone to seizures. They can be easily excited by little stimuli and are very much sensitive to excitotoxicity. Studies on Animal models have shown clearly that ECS which is equivalent of ECT in animals caused reduced number of neurons in the hippocampus and also in enterorhinal cortex.[49] Glial cell activation and also high level of neuronal plasticity after ECT is responsible for cell damage in neurons. ECT results in brain injury which is very much similar to brain insult. Angiogenesis and neurogenesis due to increased neuronal plasticity in later stages will be the result, as seen in animal studies conducted by **Madsen et al., 2005**.[50] After ECT/ECS, there is re-growth and also repair followed by activation of glial cells and proliferation triggers the process of neuro-inflammation. **Dwork et al., 2004** [51] and **Jinno and Kosaka 2008**, [52] reported that there are many morphological alterations in microglia which are a part of

hippocampus. It has also been reported that sprouting of mossy fibres in hippocampus in the post-ECT period as a probable mechanism which is responsible amnesia produced by ECT. Microglia specifically adapt to the neuroinflammatory insult and results in synaptic plasticity and also neurogenesis as reported by **Ekdahl et al., 2009**[53] and **Graeber and Streit 2010**.[54] Cholinergic systems in brain have been associated with memory. Memory and learning are closely inter-dependent on normal cholinergic neuronal transmission. Impairment in these systems has been associated to Alzheimer's disease. Acetylcholine, through Muscaranic receptor, alters the conductance of potassium in pyramidal neurons of hippocampus affecting the memory.[55]

Disruption and blood flow alterations has been suggested as one of many mechanisms of the ECT induced amnesia. It is traditionally believed that retrograde amnesia induced by ECT arises by disruption of medial temporal lobe. Some authors put forth that disruption of frontal lobe as a cause of this mechanism. They demonstrated that loss of autobiographical information due to retrograde amnesia in post ECT period is often related with increase in theta activity in the fronto-temporal region. There is a good association of sharp increase in systolic and seizure in ECT and it is also well documented. This increased blood pressure (BP) during seizure causes a "leak" in the blood-brain barrier of brain, leading to cerebral edema. This leads to the accumulation of proteins and also many macromolecules into the CSF as well as in the interstitial spaces of the brain.[56] These substances cause disturbances in

neuronal functioning, leading to cognitive impairment. The formation of memory is mediated through long-term potentiation (LTP). It has been very well demonstrated earlier that LTP is a glutamate/Ca^{2+} dependent mechanism. It is now accepted widely that LTP is a basic process by which learning and memory are mediated When seizures occurs , it leads to marked increase in levels of intracellular Ca^{2+} via the mechanism of glutamate-induced activation of NMDA receptors.[57] This causes an induction of LTP which results in saturation of neuronal system. This saturation results in the ability of further induction of LTP to be exhausted completely. Subsequently, the process of learning and also recalling is impaired. This theory was supported by the studies of **McDaniel et al., 2006**.[58] **Home RL et al., 1984** [59] have shown independently that the administration of glucocorticoid dexamethasone in humans along with ECT resulted in severe amnesia induced by ECT. **Chamberlin and Tsai, 1998**[60] demonstrated that such amnesic effect could be a result of positive feedback of glucocorticoid on NMDA receptors.

According to studies of **Andrade C et al., 2008**,[61] ECT results in cyclooxygenase-2 activity up-regulation that leads to saturation of neuronal LTP. These could be the cause for the development of retrograde amnesia. Post-seizure changes in neuronal damage have been studied and markers have been identified. These markers could help in assessment of previous neuronal injury, and help to aid in treatment. In this regard, Brain Derived Neurotrophic Factor has been described by **Toro et al., 2007**,[62] Myelin basic protein and Glial Fibrillary Acidic Protein was described earlier by **Gurnett et al.,2003**,[63]

neurofilaments as described by **Lamers et al., 2003**.[64] **Kato et al., 1982** [65] reported that the neuro specific enolase and S100b protein have been found very rarely outside the nervous system and hence they could serve as a reliable marker for neuronal change in post ECT period.

Till date understanding single mechanism of ECT is a great challenge.

There exists many controversies regarding ECT induced cognitive deficits and opposite school of thoughts.

Sackeim HA et al., [66] in the year 2007 conducted a large study with 260 patients across various centers in USA. It was a prospective study design. Cognitive outcome was analyzed by administering neuropsychological battery to all patients. The test batteries were administered before ECT, after completing the course of ECT and at follow up of 24 weeks (6 months). The batteries include.

- Modified Mini Mental State Exam scale (MMMSE),Stroop reaction time, Choice reaction time and Simple reaction time to assess psychomotor function.
- Continuous performance test (CPT) and Stroop test (ST) to assess the attention span.
- Buschke Selective Reminding Test and Complex figure test (CFT) to assess anterograde learning and memory.
- Autobiographical Memory Interview in short form (AMI-SF) was used to assess retrograde amnesia.

There was difference in cognitive measures at post ECT which was statistically significant. Seven centers showed such differences. AMI-SF and MMMSE were the ones which showed considerable differences. They suggested that adverse cognitive outcomes were produced by ECT including deficits in global cognitive function and also memory loss which may be present more than six months which may be permanent. This was mainly attributed to difference in techniques of administering ECT, waveform in which ECT is administered and type of placement of electrodes.

Effects of ECT in long term on some of memory system had been studied in group of patients suffering from bipolar disorder by **Glenda Macqueen et al., 2007**.[67] Two groups were one who did not receive ECT and another received ECT before six months. Assessment was done for visual memory, California test for verbal memory, habit memory and tasks on recollection. They found that ECT treated patients had decline in verbal learning and had some memory disturbances than controls. This difference was not caused by illness itself. They also concluded that these findings may have an impact on risk-benefit ratio of this highly effective treatment. This study also stressed the importance of addressing the cognitive effects of patients treated on ECT and to develop strategies to minimize this side effects.[67]

MacKenzie et al [68] assessed cognitive domains in the acute period within twenty four hours after administration of ECT. It was given bilaterally and the effects were observed. There were sixteen patients who were controls and were not administered ECT and thirteen patients who were given ECT.

Array of neuropsychological test batteries were administered. They observed significant variations on cancellation of letter tasks which measures attention component in ECT treated populations. The authors came to a conclusion that ECT given bilaterally may result in attention deficits in the acute period.[68]

Squire and Slater [69] reported that many patients reported memory complaints after ECT subjectively. They compared three groups with patients underwent right unilateral ECT, bitemporal ECT and no ECT . Bitemporal electrode placement resulted in more reports of memory complaints subjectively than other groups immediately after treatment but both groups reported significant memory problems than control ECT at seven month follow up leading to the suggestion that both bitemporal and right unilateral ECT placement lead to subjective memory impairment. These studies are important in the fact most of studies in ECT did not demonstrate memory impairment on standardized scales though patients had memory problems subjectively.[69]

Weeks D et al.,[70] studied a group of patients with depression, one group received ECT, the other did not receive ECT and one group was normal controls. An array of cognitive test batteries was conducted at 4th month and also at 7th month after administration of ECT. There was mild deterioration of cognitive domains at 4th month and at 7th month there was no significant difference from baseline in test batteries. It was also found that illness severity has much influence on cognitive domains. In the immediate period, within one week of administration of ECT, it was found that ECT given bilaterally had

more cognitive decline than ECT given unilaterally. But at the end of three months the two groups did not differ on their cognitive domains.[70]

Shulze-Rauschenbach et al.,[71] showed that anterograde memory problems were more for patients underwent right unilateral ECT than with repetitive transcranial magnetic stimulation (rTMS) or healthy controls who were other groups in the study. Fewer words were remembered from Rey Auditory Verbal learning test in groups who had right unilateral ECT than other groups. Retrograde amnesia was also seen in ECT patients than rTMS or control groups. Variations were found in recalling from verbal and visual stimuli given in pre ECT period. Autobiographical memories were not impaired while administering Autobiograhical Memory Interview. These studies are of view that there is impairment of retrograde memory after bitemporal electrode placement. It is less impaired in case of right unilateral ECT compared to bitemporal ECT. **McElhiney et al., 1997**[72] also concluded the same. The study conducted by **Schulze-Rauschenbach et al.,** found that there was no significant difference on any non memory cognitive measures between different groups in their study. They administered Mini-Mental Status Exam (MMSE) for assessment of overall cognitive domain, Trial making test A and B for executive functions, Weschler digit span subtest for working memory, and Word fluency test for verbal fluency and Letter number span test for assessing mental processing speed. Their study revealed the facts that memory was the one domain affected while other cognitive domains remain unaffected by right unilateral electrode placement compared to rTMS and

control population. These studies also suggested that in rTMS population memory improved from baseline since depression was treated and illness improved but reverse finding were seen in ECT treated populations, memory deteriorated from baseline value. Non memory cognitive domains were not affected according to this study.

Bifrontal placement of electrodes was done initially to avoid the involvement of hippocampal and temporal regions of brain since they are mostly concerned with memory and also in the process of learning in humans but the research was not comprehensive due to the fact concerning cognitive adverse effects and also the effectiveness of ECT conducted in this type of study design by **Sackeim et al., 2007**.[66]

Some authors argued that there will be activation of frontal lobe due to bifrontal placement of electrodes which have adverse effects on executive functions similar in fashion in which bitemporal placement of electrodes on memory by **Fattal, Crowley, Dale & Pickle, 2008**.[73] There is limited number of research and evidence on bifrontal ECT and also its various number of effects on executive and frontal lobe functions.

Around 92 patients were subjected to ECT. One group was receiving bifrontal ECT and another group was subjected to right unilateral ECT in six trials spread over a period of three weeks. There was no significant difference in MMSE scores and cognitive domains were equal for both groups and both groups were equally effective in treating depression by **Eschweiler et al.,**[74].

Bagadia VN et al., 1981[75] in the year 1981, selected two groups of patients. One belonged to schizophrenia categories who were twenty in number and another belonged to depressions that were also in same number. They were subjected to ECT and were found that there were no post ECT cognitive disturbances but some patients complained of memory disturbances subjectively.[75]

Calev et al., 1995 [76] conducted review of many studies from 1975 regarding non memory cognitive effects of ECT. They came to a conclusion that non memory cognitive adverse effects were caused by ECT in many patients. Usually cognitive functions are expected to improve after recovering from depression but they found that ECT treated patients did not have any improvement on cognitive functions taken immediately after ECT administration within few days. They also found that within days of ECT administration patients were found to be declined on their measures of visuospatial and perceptual functions, verbal fluency, intelligence testing and executive functions before and after ECT administration. They found that cognitive functions other than memory may be impaired by ECT administration though cognitive functions may be impaired by illness itself.

Another important finding of their study is that in their follow up studies after ECT administration from one week to seven months they found that nearly all measures of cognitive functions came to baseline or improved from baseline functioning . It revealed a fact that ECT induced cognitive adverse effects is of short span and temporary and improved over time. Further exploration should

be made regarding the frontal lobe functioning results and it is indecisive. The placement of electrodes in the studies of **Calev et al**. mostly had right unilateral or bilateral placement of electrodes. The differences in cognitive effects by the different electrode placement were not made in their study so it is not possible to make any conclusions on non memory cognitive impairments due to electrode placement from their study. Furthermore, regardless of differences in electrode placement ECT induced impairment in cognitive functions are of short duration according to their study.[76]

An array of test batteries had been developed for ECT and its related cognitive dysfunctions by **Vishwanath B et al.,**[77] from the NIMHANS group in 2013 (B4ECT-ReCoDe). He also derived the same conclusion that there is no significant difference in pre and post ECT period in cognitive domains.

A study was done by **Avuso-Gutierrez et al., 1982** [78] on a number of patients suffering from endogenous depression. They compared CPD-choline administered patients and patients who were on placebo after receiving bilateral ECT and within one day after application of fourth course of ECT. They used many of Weschler subscales like time taken for reorientation, Associative subtest and digit subtest, memory TEA tests. They did not take into account regarding the statistical test. No benefit was seen when CDP-choline was given for memory dysfunction induced by ECT.[78]

Abrams et al., 1967 [79] studied Schizophrenia patients who underwent unilateral non-dominant ECT thrice weekly and they were subjected to

Weschler memory scale testing. Different tests were applied each for short story telling, reproduction of verbal and visual sets, mental control, retention of paragraph and digits and orientation within hours of last session of ECT. Statistical testing were not done between groups and they also found that there was no alteration of cognitive domains between these groups.[79]

Another study was done by **Pettinati et al., 1984** [80] in depression patients. Here they compared two populations, one underwent bilateral ECT and another group was subjected to unilateral non dominant ECT. Sample size was around twenty eight patients. DSM-III was employed for diagnosis. Squire memory questionnaire was used to assess memory before and after ECT. Unilateral non-dominant ECT caused significant improvement in memory scores as opposed to bilateral ECT study design.[80]

It has been well established in many studies that ECT affects non memory cognitive functions transiently by **Calev et al.(1991a)**,[81] **Squire,L.R.(1984)**,[82] **Sackeim, H. A. (1992),**[83] It has also been observed that after ECT post-ictal disorientation and soft neurological signs may be seen.[84] Some studies have revealed that verbal fluency may be affected in acute phases after ECT by **Taylor et al.**[85]

Shapira et al, 2000 [86] studied around forty seven patients who were suffering from major depression were selected for the study. ECT was given three times per week for one group and two times with sham for another group. Cognitive status was assessed at the beginning, after twenty four hours, after

three days and at an interval of one month period. Global test battery was employed for the study. They tested for WAIS, tasks on retrograde memory, complex figure tests, orientation, tests on immediate memory, visuospatial and verbal recall, VPA and test on famous events. There was statistically significant reduction in test battery at twenty four hour period and also at three days but no difference was noted at a period of one month.[86]

Sackeim et al., 2000 [87] using Research Diagnostic Criteria, randomized control study was designed by employing eighty patients suffering from major depression. Effects of ECT given bilaterally with 1.5 times the normal threshold were compared with right unilateral ECT which was given in a threshold range of 5, 1.5 and 0.5. Results were analyzed at baseline and at a period of one week of last ECT. MMMSE, recall of pictures and recall of famous events, time taken for reorientation, BSRT, word pairs, faces, complex figure test, memory questionnaire by squire (SSMQ), AMI. There was improvement seen in all groups with time in SSMQ. All other test groups were statistically insignificant. Based on their study, a series of conclusions were arrived. Bilateral ECT fared worse followed by High dose right unilateral ECT which was followed by medium and low dose right unilateral ECT in reorientation to time test. The test was statistically significant. Other tests employed such as recall of pictures test by Randt, recall of famous events, BSRT, test for AMI, MMMSE, word pairs testing right unilateral ECT was better than bilateral ECT. In short story testing by Randt and complex figure

test, low and medium dose ECT performed better than high dose right unilateral ECT which is very much significant statistically.[87]

Lisanby et al., 2000 [88] made another comparison made between bilateral and right unilateral ECT, high doses of ECT with low doses of ECT and another group was normal controls. It was a non randomized study design. Nearly fifty five depression patients were taken up for the study. Impersonal and personal memory test, personal component of the test (PIMT-) and impersonal component of the test (PIM-I) were applied before ECT administration and at one week post scores were derived. Control population was normal while impersonal component scores deteriorated in ECT group and the results were significant statistically. The same result was derived in personal component of the test. In all these tests, bilateral ECT patients fared poorly with statistically significant results and the dose had no significant implications in these tests**.** Lisanby LH suggested that retrograde and anterograde amnesia were produced by ECT.[88]

A study was designed by **Bailine et al., 2000** [89] with depressive patients diagnosed as per DSM IV, both unipolar and bipolar depressive patients were included. Electrodes were placed in bifrontal region for one group and in bitemporal region for another group. MMSE scores were derived after application of last ECT. MMSE scores did not alter in bifrontal group but it deteriorated with significant statistical values in bitemporal group. Thus they came to a conclusion that temporal placement of electrodes resulted in more

cognitive impairment than bifrontal electrode placement which has equal efficacy but with substantially less impairment than bitemporal placement.[89]

Dubovsky et al, 2001 [90] employed Refractory depression patients diagnosed as per DSM-IV in their study. Sample size was around twenty six patients. One group were receiving placebo and another were receiving Nicardipine. They used Montogmery-Asberg rating scale for depression, Hamilton scale for depression, Beck depression inventory, MMSE and an array of test batteries for assessment of neuropsychological functions before administration of ECT, after ECT administration and after six months post ECT. They derived the results that nicardipine group had depression scores which were lower on Montogmery and Hamilton scale while Beck did not show any change immediately after ECT completion. At the six month period no change was observed between two groups. Cognitive domains deteriorated at first and then improved over six month period in both placebo and Nicardipine group. No change was observed on MMSE scores. Only significant changes were seen in two subtests on battery of neuropsychological tests. No difference was found between these two groups on cognitive domains. They inferred that adding Nicardipine to ECT may improve anti depressive effects of ECT which needs further study but it did not have any effect on cognitive effects of ECT.[90]

Tang et al 2002[91] used Piracetam in their study on ECT. A randomized study was designed which was double blind and the sample size was thirty eight which included both depression and schizophrenia patients. Patients

undergoing ECT bilaterally was subjected to placebo in one group and Piracetam was put in another group. Effects were analyzed by rating scales and also cognitive domains were assessed before ECT, after third and sixth ECT and two weeks after the administration of last ECT course. Loading dose of piracetam was 7.2 grams and 4.2 grams were used later. Results were tabulated stating that piracetam had no significant effect on ECT related cognitive and memory effects. It may have augmented ECT effects in treating illness by reducing illness scores compared to placebo group but not significant statistically.[91]

McCall et al., 2000 [92] in their studies used fixed high dose of right unilateral ECT and in another group threshold had been raised to 2.25 times the normal. Patient sample size was around seventy two and was suffering from depression. MMSE, complex figure test, Rey Auditory Verbal learning test, time taken for reorientation test, Duke and rating scales for memory had been used for the study. After the study, they found that in some tests like MMSE, reorientation time testing and Duke's test, patients subjected for fixed high dose category did not perform well relative to another group and it was significant statistically. The results in all other tests were not significant statistically.[92]

Tew et al., 2002 [93] compared high charge ECT given bilaterally with ECT given right unilaterally. Patients suffering from depression were taken up for the study. Sample size was twenty four. Elderly patients aged fifty and above were taken up for study. MMSE scores were analyzed before

administration of ECT and after ECT administration within one –three days. Patients administered ECT bilaterally had lower scores and performed poorly with scores that are significant statistically compared to other group. Around eight patients received ECT right unilaterally did poorly but the results were insignificant statistically.[93]

McCall et al., 2002[94] gave ECT right unilaterally which was around eight times than the threshold stimulus was compared with another group of ECT given bilaterally which was 1.5 times than the threshold stimulus. Sample size was forty for ECT given right unilaterally and thirty seven for ECT given bilaterally. Standardized tests were applied before administration, after 1-3 days of ECT, two and four weeks after administration of ECT. Both groups were similar in their effects to alleviate depression and causing amnesic effects which were transient. There was no difference between these groups which were significant statistically.[94]

Heikman et al., 2002 [95] studied around twenty four patients suffering from depressive disorder was diagnosed according to DSM-IV. They were spread into three groups by comparing the effects of right unilateral ECT about 400% of the threshold seizure with about 150% of threshold seizure with bifrontal ECT which was given just above threshold seizure. MMSE and Hamilton scale for depression were used for study. There was much faster response with high dose ECT which was given right unilaterally than other group. The response rate was much higher with high dose ECT than when compared to moderate dose or ECT given bifrontally.[95]

Ranjkesh et al ., 2005 [96] compared three groups of study population with a sample size of forty five cases diagnosed with depression. They were subjected to moderate dose of ECT given bifrontally, high dose of ECT given right unilaterally, low dose of ECT given bitemporally. MMSE and Hamilton scale for depression were employed for the study. Results revealed that Hamilton scale for depression did not reveal any statistical difference between these groups. MMSE revealed difference in bifrontal group compared with bitemporal and right unilateral population. They concluded their findings that all three groups were similar in their effect on treating depression but the group in which ECT given bifrontally had much less cognitive adverse effects compared with ECT given right unilaterally and bitemporally.[96]

Chanpattana et al., 2000 [97] took Schizophrenia patients in their study to compare the effects of different threshold of ECT given bilaterally. The threshold values were 4X, 2X, and 1X. Sample size was sixty two. MMSE and NST were applied before and one week after ECT. Cognitive domains did not show any significance on statistical values regarding differences between groups.[97]

Prakash et al., [98] done a study to evaluate the effect of Donepezil on cognitive domains in patients who were subjected to ECT. It was a triple blind study conducted on 45 patients. They were separated into two groups, one receiving donepezil and the other receiving placebo. The patients were analyzed before and after administration of ECT. Cognitive domains and recovery of patients with respect to cognitive domains were analyzed. Results

were tabulated. They found that patients receiving donepezil recovered faster than patients receiving placebo on various aspects of cognitive domains in post-ECT period. They concluded that this faster recovery time with donepezil has therapeutic implications on cognitive deficits in post-ECT period immediately.[98]

The major indication of ECT is development of resistance overtime to medications. There is also a trend to stop medications before administration of ECT. A study was undertaken by **Sackeim et al., 2009** [99] to compare patients receiving placebo and concomitant medications along with ECT (Nortriptline or Venlafaxine) and also studied the effects of high dose ECT given right unilaterally with moderate dose ECT given bilaterally. Sample size was three nineteen, with diagnosis of depression. Hamilton scale for depression rating was used. They derived the results that adding Nortriptiline to ECT resulted in more efficacious treatment and also resulted in less cognitive side effects than the addition of placebo. But Venlafaxine improved the effects of ECT only mildly but also resulted in deterioration of cognitive domains. They also found that ECT given right unilaterally in high doses resulted in efficacy of treatment similar or above than ECT given bilaterally in medium doses with lesser adverse effects on cognitive domains compared to ECT given bilaterally.[99]

A comparative study was done by **Geretsegger et al.,** [100] between methohexital and propofol as anesthetic agents for treating patients with the help of ECT. It was a double blind randomized study. They compared the quality of seizures, efficacy of response and performance on various aspects of

cognition. Patients sample size was around 50 and were diagnosed with depressive disorder. One group received propofol and the other received methohexital. Duration of seizures, frequency of pulse, and efficacy of seizure index, diastolic and systolic blood pressure and also post ictal suppression were measured. They were seen before ECT, after 3-5 ECT, after completion of ECT course, follow-up at 2 and also 8 weeks. Propofol patients' quality of seizures were shorter, blood pressure was increased mildly in post-ECT period when compared to the other group. They concluded their findings that propofol is similar in illness improvement but cognitive improvement is better than methohexital with only significant results in two trials. So propofol was considered a better anesthetic agent for patients receiving ECT.[100]

Thirty nine patients older than 60 years were taken up for study by **Stoppe et al 2006**.[101] Their diagnosis were depression and they were subjected to fixed, high doses of ECT given bilaterally and unilaterally (right unilateral). Domains on cognition and severity of illness were tested before ECT administration, during and after one month of treatment. Results turned out to be same for RUL ECT and bilateral ECT in terms of rates of remission. Illness reduction was same in both groups but in terms of cognitive domain ECT given bilaterally had poorer outcome compared with other group. Conclusions were arrived that both groups were same in reducing the severity of illness but bilateral ECT had more cognitive adverse effects compared with RUL ECT.[101]

Mohan et al.[102] compared two groups undergoing ECT bilaterally, one group receiving intensity of stimulus just above threshold of seizure and other

receiving 2.5 times above threshold of seizure were compared to assess the rate of recovery in manic patients. The effects on cognition were also assessed with MMSE. Both groups improved significantly after the treatment course with no difference between them. Both type of interventions proved to be safe and efficacious with no difference between them statistically.[102]

Acute manic patients were assessed by **Hiremani et al** [103] by giving ECT bifrontally and bitemporally with regards to response for treatment and cognitive outcomes of the two different electrode placements. Sample size was thirty six diagnosed as mania as per DSM-IV criteria. Mood stabilizers were not used during the study. YMRS, MMSE, Fluency testing, CFT, learning test on paired associate groups and one part of trial making test were employed for the study. They concluded that patients who were on treatment with bifrontal ECT showed faster recovery when compared to ECT given bitemporally. Cognitive domains revealed no difference between the groups significantly.[103]

ECT given bifrontally in moderate dose was compared with ECT given bitemporally with low dose by **Barekatain et al.,**[104] Sample size was 28 patients who were diagnosed with manic disorder. MMSE scores and standardized rating scale for mania (YMRS) were employed for the study. A course of 6 ECTs were given. There was no difference in scores between the groups before the administration of ECT. But MMSE scoring of the two groups varied significantly after a course of 6 ECTs. ECTs given bifrontally was as efficacious as ECT given bitemporally with lesser cognitive adverse effects in manic disorder patients.[104]

There was a randomized study by **Smith et al** [105] in patients suffering from unipolar depression to compare two groups to analyze the effects on memory between continuation of ECT and continuation of drugs (lithium and nortriptyline). Sample size was eighty five and RAVLT, AMI, assessment of subjective memory were all employed at twelve weeks and twenty four weeks after ECT in addition to baseline assessment. There was statistically significant difference on AMI at twelve weeks from baseline in drug group. All others were insignificant. There was improvement in memory scoring from twelve to twenty four weeks suggesting recovery of deficits in anterograde memory induced by ECT. There were no significant changes in outcomes comparing both groups.[105]

Effects of cognition in bifrontal and right unilateral with pulses in ultra brief duration were compared by **Sienaert et al**.[106] Sixty four depressive patients were taken up for study. One group received ECT bifrontally at 1.5 times the threshold of seizure and another right unilaterally at 6 times the threshold. Assessment was made at one week and also at six weeks after ECT administration. Cognitive domains were normal in both groups. Memory was found to be improved in some patients. Conclusion was arrived that both techniques does not have adverse effect on cognition.[106]

Around fifteen patients suffering from depression older than forty five years were subjected to bitemporal ECT by **Warnell et al**.[107] One group were interrupted in the post seizure period by infusing propofol and another group by placebo. One to three days later subscales on WMS were measured like

retention of paragraph, orientation, digits, verbal, mental control and short story. There was significant delay in auditory and immediate memory. Other tests revealed insignificant results. They came to a conclusion that interruption of post seizure period by infusing propofol resulted in decreased cognitive adverse effects after ECT which may be clinically important.[107]

Kellner et al [108] studied bifrontal ECT, ECT given bitemporally and right unilaterally in depressive population in a multi centered trial which was a double blind and randomized trial.. Two thirty patients were studied. All were good in reducing illness severity. ECT given bitemporally had an edge in reducing illness severity rapidly than other groups. Cognitive outcomes were not significant statistically between groups. They also suggested that bifrontal has similar cognitive outcome as that of bitemporal ECT in contrast to other studies.[108]

Two groups were chosen by **Sackeim HA et al.,**[109] and were given ECT bilaterally or right unilaterally at high dosage of stimulus (supra threshold) or low dosage of stimulus. Sample size was 71 patients suffering from depression. At the end of treatment, after return of orientation, 3 tasks on visual cancellations were given immediately following ECT. Omission errors were calculated. Intensity of stimulus and type of placement of electrodes were noted. They found that ECT given right unilaterally with high intensity resulted in neglect of cancellation forms on left side, while asymmetry was noted in ECT given in low intensity. On the other hand, ECT given bilaterally in high dosage resulted in greater omission in the cancellation forms on right side.

They concluded that when the intensity of stimulus is raised in patients receiving ECT unilaterally, the functions of the right hemisphere are more affected.[109]

A review of studies was done by **Verwijk E et al.,** [110] about ECT given right unilaterally in ultra brief pulse and brief pulse wave forms till 2011 were undertaken. The effects were analyzed immediately within 1-7 days, 1-6 months and after six months after administration of ECT. There was impairment noted in verbal fluency, memory of personal information and also anterograde memory in the immediate aftermath of ECT. There was also decline in the processing speed and working memory. There was normalization in anterograde memory and verbal fluency at 1-6 months period but autobiographical memory was the one which persisted but somewhat improved. They concluded that ECT given in ultra brief pulse waveform showed less deterioration in autobiographical memory than brief pulse waveforms. All other effects on cognitive domains appear to be transient and resolve over time.[110]

Cognitive effects in adolescents undergoing ECT were assessed by **Neera Ghaziuddin et al.,** [111]. They also assessed whether any impairments will persist many months following ECT. Sample size was sixteen adolescents diagnosed with symptoms of mood disorder. Cognitive domains were assessed before, immediately after ECT and months after receiving ECT. They found that there was deficits in attention, concentration, delayed recall in verbal and visual components and also in component of verbal fluency. There was no deficit in the area of ability to solve new problems initially. At months after

assessing these deficits were found to be normalized or even improved. They concluded that ECT does not cause any long term cognitive deficits and all the effects are transient and short lived in immediate period.[111]

Cognitive domains were assessed immediately after a single session of ECT by **Rami et al**., [112] A controlled study had been undertaken with a sample size of 24 patients who were subjected to ECT as maintenance treatment. They were spread as control and experimental group. Effects on visuospatial ability, attention processing, frontal and learning functions were made out. Most of the cognitive domains remain unaltered after a session of single ECT but there was deterioration on visuospatial ability on experimental group after ECT implicating that ECT may be responsible for acute effects on cognitive domains of right hemisphere.[112]

Ng C et al., [113] studied ECT given right unilaterally at 2.5 times the seizure threshold with a sample size of 32 patients diagnosed with depression. Assessment was made with an array of tests like tests for personal memory Wechsler intelligent scale-short version, Randt test for memory, self rating of memory were given at the baseline during the treatment course and after one month of administration of ECT. There was deficit in anterograde memory in the short term period. There was decrease in mean scores in tests for personal memory and also in Randt test for memory of 32.5% and 14.8% respectively after the course of ECT. All the deficits improved significantly after one month. There was no deficit of non-memory cognitive domain. They concluded that ECT given right unilaterally does not result in significant or permanent

cognitive impairments, but the effect of treatment is insufficient in clinical settings.[113]

There is some consensus that ECT results in rise of blood pressure levels which may result in the breakdown and breach of blood-brain barrier. A study was done by **Hamilton et al.,** [114] with a sample size of 27 patients who were diagnosed with depression. Cognitive domains were analyzed with Benton test before and 3 hours after administration of ECT. They found that deterioration in cognitive domains were correlated significantly when the systolic blood pressure raised significantly during the course of treatment.[114]

Squire L R reviewed many studies which clarify the nature and the type of memory deficits associated with ECT. He also found that ECT given bilaterally resulted in significant loss of anterograde memory than ECT given right unilaterally. Bilateral ECT also resulted in significant amnesia in retrograde fashion than ECT given unilaterally. Memory reactivation just before administration of ECT did not result in any type of amnesia. Ability to learn things newly were maintained in long term periods after administration of ECT but subjective memory complaints is a common feature in patients who received ECT bilaterally. Since both treatments are equally efficacious, ECT given right unilaterally maybe preferred to ECT given bilaterally, because the adverse effects on memory is much lesser.

Nearly a quarter of patients suffering from dementia also have comorbid depression. Nearly one third of them require ECT as a treatment, since they do

not recover with anti-depressants. But, the major concern is ECT's effect on cognitive domains and also memory in patients with dementia. 31 patients suffering from dementia with depression were administered ECT by **Rao V et al.,** [115] MMSE and Montgomery-Asberg scale for depression are employed to assess cognitive domains and illness severity respectively. Among the 31 patients majority had dementia of vascular origin, the next in order was degenerative dementia and finally Alzheimer's dementia. There was a statistically significant decline in illness severity which was evident in rating scale for depression. Delirium has developed in half of the patients. There was also a statistically significant improvement in cognitive domains as evidenced by MMSE scores. They concluded that ECT can be used as an effective treatment option for patients suffering from dementia with depression which leads to global improvements in both cognitive domains and mood. But multiple treatments may be an option to achieve this effect.[115]

Sackeim et al.,[116] selected around 90 patients suffering from depression using criteria based on research diagnostics and studied the effects of ECT given bilaterally at 2.5 times the threshold of seizure with ECT given right unilaterally at 6 times the threshold of seizure either with brief pulse waveforms at 1.5ms or ultra brief pulse waveforms at 0.3ms. Effects were made out at before administration, immediate period, after one week and also at two and six months. Tests conducted include time taken for reorientation, recognition of sentence, BSRT, MMSE, test to assess cancellation task, verbal fluency, recall of words and recognition, recall of stories, recognition of

shapes, CFT, AMI, recognition of neutral faces. Most of domains declined at one week period with statistically significant values. Brief pulse ECT had poorer outcomes when compared to ECT given in ultra brief pulse form. Performance on cancellation tasks, recognition of words and recall, verbal fluency, recognition of faces were declined in brief pulse than ultra brief pulse ECT in acute stages with values that were significant statistically. Values on CFT, MMSE and also BSRT reveal poor scores on brief pulse when compared to ultra brief pulse at one week period. Recall of stories, AMI and rating of memory by patients were all declined more with brief pulse than other group at one week period. They concluded that ECT given right unilaterally in ultra brief pulse had less cognitive decline than other groups. Bilateral ECT had more decline on cognitive domains at one week period and also more than ECT given right unilaterally.[116]

The American agency named **Food and drug administration (FDA), 2011** [117] prepared a detailed and exclusive document for assessing the safety profile of the ECT machines. The effect of ECT on memory and cognition is studied in detail in the summary document by using various literatures. Four systematic reviews and meta-analyses were found during their study.

The following conclusions were obtained from their reviews:

- Deficits in memory were evident through various literatures.
- Immediately after the application of ECT or at the end of treatment considerable deficits in cognition and memory occurred.

- The common type of retrograde memory lost after ECT is autobiographical memory
- Three to four fifth of the patients have such memory loss.
- Sine wave ECT has a higher impact on memory than brief pulse stimulation ECT.
- The risk of memory impairment after ECT is linked to the placement of electrodes on the dominant brain hemisphere and both the hemispheres.
- The energy dose of ECT greatly affects the degree of memory deficits.
- Application of electrical stimulus higher than the patient's seizure threshold resulted in better results, but increases the risk of memory impairment in unilateral ECT.
- In majority of the patients the memory deficits remaining after 6 months is not evident from various clinical trials.
- The effect of ECT on the patients is not altered by the type of mental illness they are suffering from.
- The maximum efficacy of ECT can only be gained by taking a greater risk in the deficits of memory.
- The conclusions are depending on intra-personal and inter-personal.

- The quality of life is affected by this type of memory deficits in the patients.
- Comparing the various studies which are performed in this topic has a lot of methodology issues.

According to NICE guidelines[118] the time period is divided into the following depending on the time duration:

- **Immediate post-ECT effects**: this time period includes the first 24 hours immediately after the termination of ECT seizure during which the effects are more acute.
- **Sub-acute post-ECT effects**: this time period extends beyond 24 hours and less than 2 weeks after the final termination of ECT seizure during which the ECT effects starts or persists.
- **Medium-term effects**: this time period extends beyond 2 weeks and less than 3 months after receiving the last ECT course during which the ECT effects may start or persist.
- **Longer-term effects**: this time period extends beyond 3 months and less than 6 months after the final course of ECT during which the ECT effects begins or exists.
- **Long term effects**: this time period extends 6 months after the last course of ECT during which the ECT effects begins or exists.

The FDA identified the following using the studies in literature from the Global Cognitive Function applying the mini-MMES:

- The immediate effect of ECT after the treatment is higher when ECT is applied bilaterally than when ECT is applied unilaterally is found to be proved only by a small amount of evidence.
- There is no clear evidence in the change of global executive function. The existing literatures are also inconclusive and contradictory.
- Very little evidences are available to prove that placing electrodes bitemporally is poorer that placing electrodes bifrontally in the sub-acute period after applying ECT. These evidences are also found to be ambivalent with respect to electrode placement, the energy difference and with respect to change from the global cognitive function baseline.
- It has been found that there are no considerable global cognitive function differences between frontal ECT application and unilateral ECT and bilateral ECT application of ultra-brief pulse in the medium time period.
- In the “longer-term effects” post-ECT, There are only little evidences showing that there is small baseline global cognitive function deviation from the baseline assessment in the longer term period.
- These systematic reviews and meta-analyses also found that placing electrodes bilaterally causes a 6 to 10 percentage lesser score of MMSE than lacing electrodes unilaterally immediately after the application of ECT. There was also no statistical difference in MMSE scores when

medium energy and unilaterally placed electrode ECT was compared against low energy and unilaterally placed electrode ECT. Similarly no differences in MMSE scores were found when the placing electrode bilaterally was compared for low energy, medium energy and high energy. But, a small difference still existed in these conditions. This increased considerably until 2 months after ECT.

- When patients were administered bilateral ECT and /or high dosage of energy in ECT, they had a twelve percent decrease in the score of MMSE when compared to the unilateral ECT and/or low dosage of energy in ECT.

.

AIMS AND OBJECTIVES

AIM

The aim of present study is to assess, evaluate and compare the cognitive functions of psychiatric patients prior to and after electroconvulsive therapy administration.

OBJECTIVES

1. To study the cognitive functions before, immediately after first electroconvulsive therapy and at the end of one week of last electroconvulsive therapy.
2. To compare the cognitive functions before and after electroconvulsive therapy administration.
3. To correlate the cognitive functions and illness variable with electroconvulsive therapy.

HYPOTHESIS

Null hypothesis:

There is no change in cognitive functions following electroconvulsive therapy.

Alternate hypothesis:

There is significant change in cognitive functions following electroconvulsive therapy.

INCLUSION CRITERIA

1) Patients suffering psychiatric illness and planned for electroconvulsive therapy as recommended treatment option.
2) Age 18-55 years of both sexes.
3) Had not subjected to Electroconvulsive therapy within past 6 months.
4) Had given consent to participate in the study.

EXCLUSION CRITERIA

1) Patients with history of co-morbid Axis I psychiatric illness.
2) Patients having history of physical illness and neurological disorder.
3) History of previous ECT within six months period.
4) Patients who are diagnosed as mentally retarded, dementia, substance induced and organic psychosis.
5) History of head injury in the past which is severe enough to cause loss of memory and cognitive decline.
6) Patients having learning disability.
7) Patients not consented for the study.

MATERIALS AND METHODOLOGY

TOOLS USED

1) Semi-structured Proforma
2) PGI memory test
3) Digit symbol substitution test
4) Color Trial test-1
5) Color Trial test-2
6) Controlled Oral Word Association test (COWA)
7) Addenbrooke's Cognitive rating scale (ACE-R)

PGI MEMORY TEST

The Post Graduate Institute, Chandigarh, memory scale had been designed by Pershad and Wig.[119] Memory is generally attributed as a temporal lobe function. Temporal lobe on right side is responsible for non-verbal component of memory and left side to the verbal component of memory. This scale provides a simple and very comprehensive technique to assess non verbal and verbal memories on the basis of neurological theory. Long term, short term and very short term memories are assessed on the basis of evidences experimentally and immediate, recent and remote memories on the basis of clinical practice setting on evaluation of memory. It is specially designed for Indian population. There are ten subtests to assess different components in memory such as remote memory, recent memory, mental balance, attention and

concentration, delayed recall, immediate recall, verbal retention for similar pairs, verbal retention for dissimilar pairs, visual retention and visual recognition of some common objects. This test has been well acclaimed and validated with international tests like Weschler test for adult intelligence and also Boston memory scale. Administration test takes around fifteen minutes.

DIGIT SYMBOL SUBSTITUTION TEST

Digit symbol substitution test is a sub test of Weschler Adult Intelligence scale.[120] It is timed and fine motor test and very much sensitive for brain damage, psychiatric and other non neurological problems. It assess the mental speed and also sustained attention, visuomotor coordination, response speed and motor persistence. Information processing must be rapid to substitute symbol for digits quickly and accurately. It contains a sheet of paper in which number from 1-9 are arranged randomly in four rows each containing 25 square boxes. The subject must do substitution of symbols for digits by using the number-symbol key given at the top of sheet. The subject must do the test as fast as possible after a practice test and time taken for the test is noted. The test usually takes about seven minutes. It is also called digit symbol in WAIS-R, Digit-Symbol-Coding in WAIS-III and Coding in WAIS-IV.

COLOR TRIAL TEST

This test was created originally by D'Elia, Satz, Uchiyama and White in 1996 [121] and adopted by WHO as one part of a multicenter study regarding HIV infection. It is a derivative of Trial making test and it does not contain alphabets and free from language. It consists of two parts. It measures focused attention. In addition PART 1 measures simple sequencing, sustained attention and perceptual tracking. PART 2 also measures the ability of mental flexibility in addition to the above mentioned tasks. It measures focused attention because in both parts subjects must ignore the irrelevant numbers and scan for the next number in their sequence. In PART 1 of the test, numbers from 1-25 are spread randomly in a sheet of paper with pink circles for odd number and yellow circles for even number. The subjects must point and connect number from 1-25 in ascending order. In PART 2 of the test , numbers from 2-25 are printed twice once on yellow circles and once on pink circles. In this test subjects are asked to point the numbers in ascending order but with alternating colors. It has main part and practice test sheet, main parts of color trial are given only after the subject has understood the principle and performed satisfactorily in practice sheet. Subjects are instructed to do the test as fast as possible and time taken is noted down. The test takes about ten minutes.

CONTROLLED ORAL WORD ASSOCIATION TEST

It was previously called as Verbal associative fluency test and now it has been changed to COWA test. It is a part of Halstead-Reitan neuropsychological test batteries. It measures the verbal fluency and used to assess executive functions. Verbal fluency is the capacity of subjects to generate as many new words as possible in a manner which is regulated. The new words can be generated according to words starting with specific letters or specific category, while the former is COWA test and latter is Category test. COWA test was designed by Benton and Hamsher in 1989.[122] COWA test assess the phonemic fluency. In this test subject is asked to name as many new words as possible starting with consonants F, A, S. Subjects are instructed not to repeat the same words, give the names of person or places. Subjects are also asked to avoid proper nouns and giving different suffixes for the same word. If person does not know English, they are asked to name with consonants starting from Ka, Pa, Ma. A practice test is given with letter other than the indicated letters before main test. They are given a three trial with each consonant for one minute with a gap in between the trials and the new words generated are noted down. The average new words generated in three trials forms the score. This test takes about five minutes.

ADDENBROOKE'S COGNITIVE EXAMINATION-REVISED

It is a neuropsychological test and it is very brief and used for the assessment of overall cognitive functions. It is a theoretically motivated

component of MMSE. It is used as a bedside test to assess cognition. It has good sensitivity and specificity to identify dementia and also has very good patient acceptability. It is very useful to demarcate mild cognitive impairment. It is also good to differentiate cognitive decline caused from depression with dementia. It also delineates Alzheimer dementia with fronto temporal dementia.[123] It expands the scope of MMSE which has an overall score of 30. ACE-R tests five domains namely orientation and attention, memory, fluency, language and visuospatial abilities. A score of 18 for orientation and attention, 26 for memory component, 14 for fluency, 26 for language component and 16 for abilities on visuospatial functions is given. The total score for ACE-R test is 100. The test takes around fifteen minutes.

OPERATIONAL DESIGN

This study is a hospital based study which has been conducted at Government Medical College Hospital, Tirunelveli. It is a prospective study design conducted for a period of one and half years. Approval from Institutional Ethical Committee, Government medical college hospital, Tirunelveli had been obtained

The samples selected in the study were patients attending the psychiatry department and who were scheduled for Electro convulsive therapy. The diagnoses of the condition of patients were performed with ICD-10 criteria.

After the Institutional Ethical Committee approval had been obtained, patients scheduled for Electroconvulsive therapy in the Department of Psychiatry,

Government medical college, Tirunelveli were recruited for the current study. Informed consent had been obtained. Informed consent in Tamil which had been approved earlier by the Institutional Ethical Committee Review Board had been used to obtain consent from the participating patients.

After obtaining consent, patients who meet the exclusion and inclusion criteria were enrolled in the study design. Patients taken up for the study were able to understand the nature and purpose of the study. Uncooperative patients and patients with acute psychosis were ruled out from the study.

Semi-structured proforma were administered for the patients taken up for the study. Socio-demographic profile as per the proforma was collected. Complete general physical examination and also detailed neurological evaluation were done before the study. All the subjects underwent cognitive assessment which lasted around 90 minutes. All the tests were carried out in a fixed order according to standardized administration procedure in a quiet room.

All the patients underwent the tests in the following phases:

-Baseline: Within 24 hours before administration of ECT.

-Post 1st ECT: Within 24 hours after administration of 1st session of ECT.

-Post –ECT: One week after administration of last session of ECT.

Electroconvulsive therapy is administered using the standard ECT machine with a Brief pulse of 60 Hz, 800mAmp and Pulse width of 1.2 ms for a total duration of 1 second bifrontotemporally to all the subjects. The

treatment process is carried out for all patients are carried out thrice weekly in a total of six sittings, unless otherwise specified. General Anesthesia with proper and adequate pre-anesthetic medications was given after which patients underwent Electroconvulsive therapy.

The results were collected, tabulated and were analyzed.

STATISTICAL DESIGN

The results collected were compared using statistical analysis. Repeated measures ANOVA also called as correlated ANOVA is used to compare the same subjects who were measured at baseline, after first ECT and one week post ECT period. Post- Hoc analysis using Fisher's test with least significant difference was used to compare inter groups and their significance was measured after ANOVA testing. To compare the mean difference between two different groups (Male and Female) t-test was used. The F values and p values which measure the significance of the test have been obtained and tabulated. As per statistical design, 95 % confidence interval was set with associated p values< 0.05 is significant. Thus the test is significant if p value is less than 0.05 according to statistical design.

RESULTS

Table-1

Table showing the Socio-Demographic Profile of Patients

S.No	Variable		Number of Patients (n=36)	Percentage (%)
1	Sex	Male	23	63.9
		Female	13	36.1
2	Age	18 - 25	8	22.2
		26 – 40	25	69.5
		41 - 55	3	8.3
3	Domicile	Rural	26	72.2
		Urban	10	27.8
4	Socio-Economic Status	Low	18	50
		Middle	14	38.9
		High	4	11.1
5	Marital Status	Married	20	55.6
		Single	16	44.4
6	Education	Primary	11	30.6
		Secondary	20	55.6
		Graduate	5	13.8
7	Diagnosis	Mania	8	22.2
		Schizophrenia	16	44.5
		Depression	12	33.3

Table 1 shows the socio-demographic details of patients who underwent the current study. Males in the study were 23 in number which constituted 63.9% of study population and females 13 in number which made up 36.1% of population. Most common age group of people were 26-40 yrs which made up 69.5% of population followed by 18-25 yrs and then least people were in 41-55 yrs age group constituting only 8.3% of population. Most people belonged to rural category constituting around 72.2% and urban people made up 27.8% of population. Low socioeconomic status was seen in majority of population constituting 50% of study, followed by middle and high groups around 38.9% and 11.1% respectively. Married population and unmarried people were 20 and 16 in number respectively. In the current study none of the patients were completely illiterate, 11.6% of population at least had primary education and 55.6% of population had secondary education (>5^{th} STD) and only 13.8% (5 in number) were graduated. Schizophrenia patients constituted the majority of study population comprising of 44.5% of population (16) followed by Depression (12) comprising of 33.3% of population and Mania (8) constituting 22.2 % of study population.

Table-2

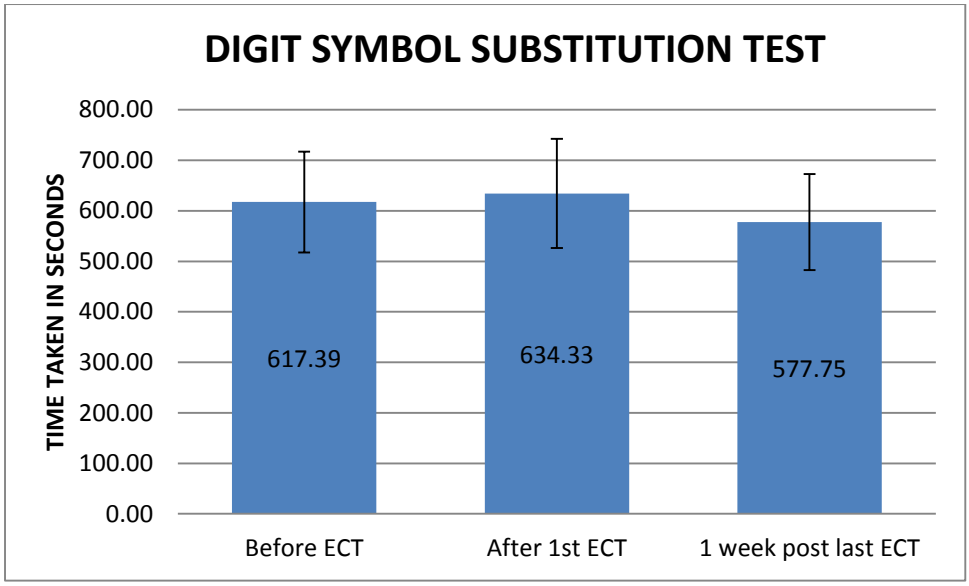
Table showing the Mean, Standard Deviation and Repeated Measures ANOVA Scores of Digit Symbol Substitution Test

Groups (n=36)	Mean	S.D	Statistics	
			F	P
Before ECT	617.39	99.61	46.64	<0.0001
After 1st ECT	634.33	107.87		
One week post last ECT	577.75	95.08		

Table-3

Table showing Inter-group comparisons using Fisher's Post-Hoc Test in Digit Symbol Substitution Test

S.No	Comparison of Groups	Statistics
1	Before ECT and After 1st ECT	p=0.003
2	After 1st ECT and One week post last ECT	p<0.0001
3	Before ECT and One week post last ECT	p<0.0001



Graph showing the Mean and Standard Deviation of Digit Symbol Substitution Test

The mean scores and standard deviation obtained in Digit symbol substitution test were tabulated in Table 2. ANOVA test applied revealed that there was significant variation between the baseline, after first ECT and one week post completion of ECT. The difference was highly significant statistically. ($F=46.64$)($p<0.0001$)

Table 3 shows the inter group comparison between different phases on Digit symbol substitution test. Post Hoc test using Fisher test after ANOVA testing revealed that there was significant difference between baseline and first ECT. ($p=0.003$) and the difference between first ECT and 1week post ECT and baseline and 1week post ECT were highly significant statistically. ($p<0.0001$)

Table-4

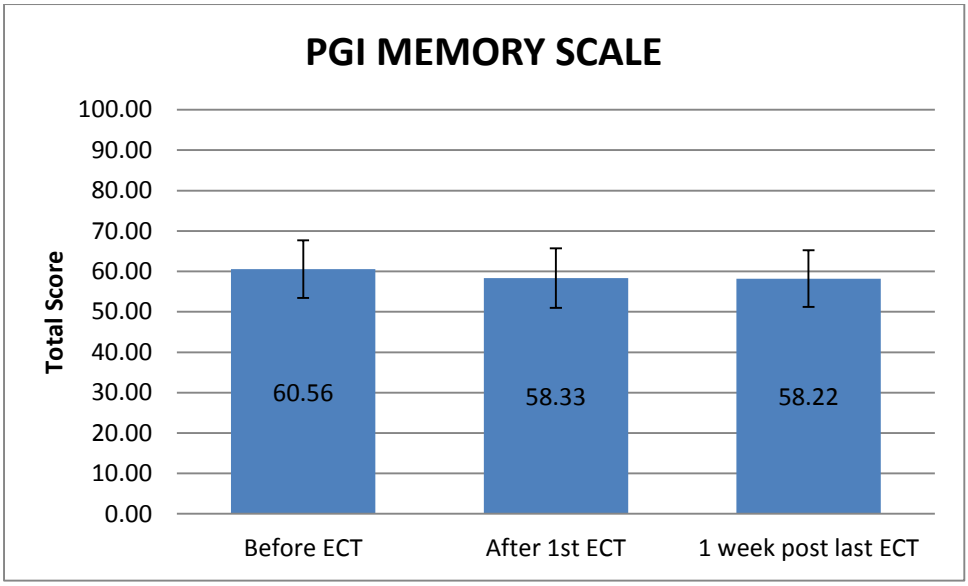
Table showing the Mean, Standard Deviation and Repeated Measures ANOVA Scores of PGI Memory Scale

Groups (n=36)	Mean	S.D	Statistics	
			F	P
Before ECT	60.56	7.12	13.62	<0.0001
After 1st ECT	58.33	7.38		
One week post last ECT	58.22	7.01		

Table-5

Table showing Inter-group comparisons using Fisher's Post-Hoc Test in PGI Memory Scale

S.No	Comparison of Groups	Statistics
1	Before ECT and After 1st ECT	p<0.0001
2	After 1st ECT and One week post last ECT	p=0.842
3	Before ECT and One week post last ECT	p<0.0001



Graph showing the Mean and Standard Deviation of PGI Memory Scale

The mean scores and standard deviation obtained in PGI memory scale were tabulated in Table 4. ANOVA test applied revealed that there was significant variation between the baseline, after first ECT and one week post completion of ECT. The difference was highly significant statistically. ($F=13.62$) ($p<0.0001$)

Table 5 shows the inter group comparison between different phases on PGI memory scale. Post Hoc test using Fisher test after ANOVA testing revealed that there was significant difference between baseline and first ECT. ($p<0.0001$) and the difference between first ECT and 1week post ECT was not significant statistically ($p=0.842$) and the difference between baseline and 1 week post ECT was highly significant ($p<0.0001$)

Table-6

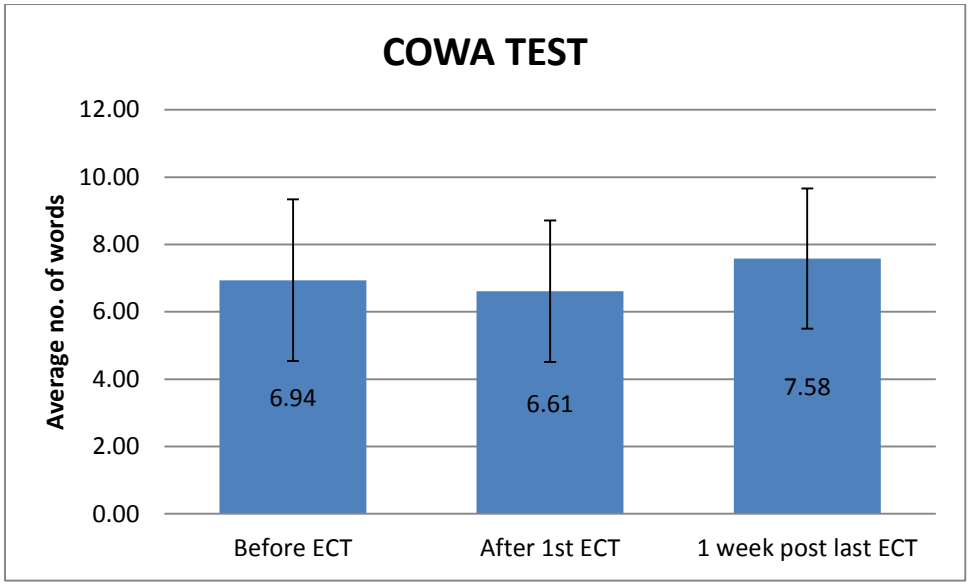
Table showing the Mean, Standard Deviation and Repeated Measures ANOVA Scores of COWA Test

Groups (n=36)	Mean	S.D	Statistics	
			F	P
Before ECT	6.94	2.40	16.58	<0.0001
After 1st ECT	6.61	2.10		
One week post last ECT	7.58	2.08		

Table-7

Table showing Inter-group comparisons using Fisher's Post-Hoc Test in COWA Test

S.No	Comparison of Groups	Statistics
1	Before ECT and After 1st ECT	p=0.003
2	After 1st ECT and One week post last ECT	p<0.0001
3	Before ECT and One week post last ECT	p<0.0001



Graph showing the Mean and Standard Deviation of COWA Test

The mean scores and standard deviation obtained in Controlled Oral Word Association Test were tabulated in Table 6. ANOVA test applied revealed that there was significant variation between the baseline, after first ECT and one week post completion of ECT. The difference was highly significant statistically. ($F=16.58$) ($p<0.0001$)

Table 7 shows the inter group comparison between different phases on Controlled Oral Word Association test. Post Hoc test using Fisher test after ANOVA testing revealed that there was no significant difference between baseline and first ECT. ($p=0.05$) and the difference between first ECT and 1week post ECT was highly significant statistically ($p<0.0001$) and the difference between baseline and 1 week post ECT was significant ($p=0.001$).

Table-8

Table showing the Mean, Standard Deviation and Repeated Measures ANOVA Scores of Color Trial Test 1

Groups (n=36)	Mean	S.D	Statistics	
			F	P
Before ECT	195.89	70.56	42.01	<0.0001
After 1st ECT	210.03	78.01		
One week post last ECT	167.94	67.18		

Table-9

Table showing Inter-group comparisons using Fisher's Post-Hoc Test in Color Trial Test 1

S.No	Comparison of Groups	Statistics
1	Before ECT and After 1st ECT	p=0.003
2	After 1st ECT and One week post last ECT	p<0.0001
3	Before ECT and One week post last ECT	p<0.0001

Table-10

Table showing the Mean, Standard Deviation and Repeated Measures ANOVA Scores of Color Trial Test 2

Groups (n=36)	Mean	S.D	Statistics	
			F	P
Before ECT	396.72	105.53	41.83	<0.0001
After 1st ECT	421.42	117.73		
One week post last ECT	356.19	107.43		

Table-11

Table showing Inter-group comparisons using Fisher's Post-Hoc Test in Color Trial Test 2

S.No	Comparison of Groups	Statistics
1	Before ECT and After 1st ECT	p=0.0010
2	After 1st ECT and One week post last ECT	p<0.0001
3	Before ECT and One week post last ECT	p<0.0001

Table-12

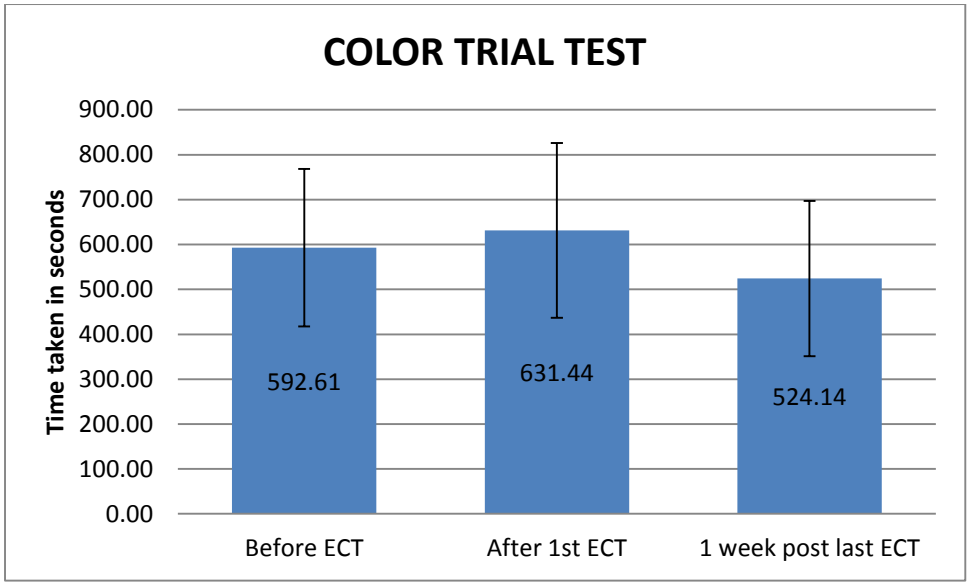
Table showing the Mean, Standard Deviation and Repeated Measures ANOVA Scores of Color Trial Test Total

Groups (n=36)	Mean	S.D	Statistics	
			F	P
Before ECT	592.61	175.44	45.68	<0.0001
After 1st ECT	631.44	194.89		
One week post last ECT	524.14	172.67		

Table-13

Table showing Inter-group comparisons using Fisher's Post-Hoc Test in Color Trial Test Total

S.No	Comparison of Groups	Statistics
1	Before ECT and After 1st ECT	P=0.001
2	After 1st ECT and One week post last ECT	p<0.0001
3	Before ECT and One week post last ECT	p<0.0001



Graph showing the Mean and Standard Deviation of Color Trial Test

The mean scores and standard deviation obtained in Color Trial Test Total score was tabulated in Table 12. ANOVA test applied revealed that there was significant variation between the baseline, after first ECT and one week post completion of ECT. The difference was highly significant statistically. ($F=45.68$, $p<0.0001$).

Table 13 shows the inter group comparison between different phases on Color Trial Test Total Score. Post Hoc test using Fisher test after ANOVA testing revealed that there was significant difference between baseline and first ECT. ($p=0.001$) and the difference between first ECT and 1week post ECT was highly significant statistically ($p<0.0001$) and the difference between baseline and 1 week post ECT was highly significant ($p<0.0001$).

Table-14

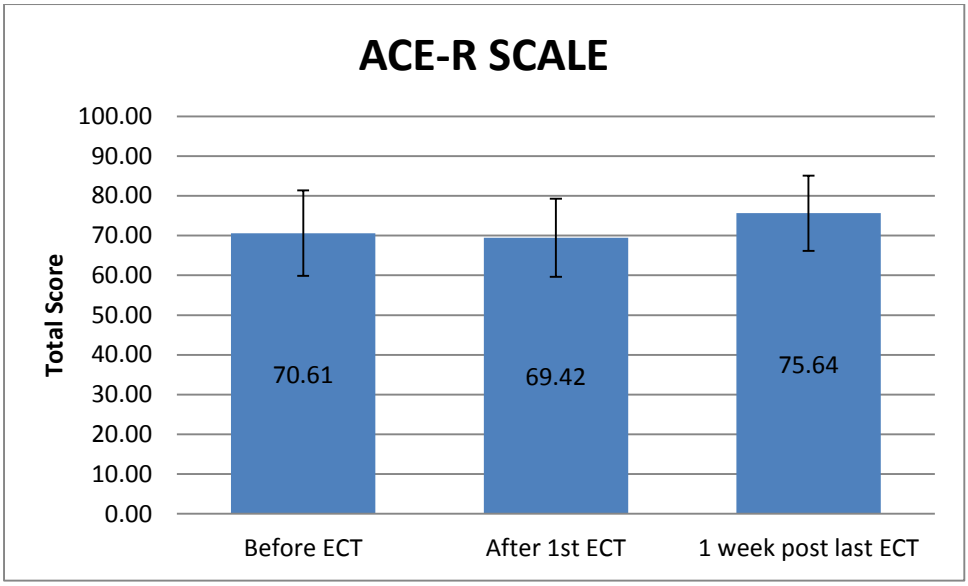
Table showing the Mean, Standard Deviation and Repeated Measures ANOVA Scores of ACE-R Test

	Mean	S.D	Statistics	
			F	P
Before ECT	70.61	10.74	82.53	<0.0001
After 1st ECT	69.42	9.83		
One week post last ECT	75.64	9.46		

Table-15

Table showing Inter-group comparisons using Fisher's Post-Hoc Test in ACE-R Test

S.No	Comparison of Groups	Statistics
1	Before ECT and After 1st ECT	p=0.023
2	After 1st ECT and One week post last ECT	p<0.0001
3	Before ECT and One week post last ECT	p<0.0001



Graph showing the Mean and Standard Deviation of ACE-R Test

The mean scores and standard deviation obtained in ACE-R scale were tabulated in Table 14. ANOVA test applied revealed that there was significant variation between the baseline, after first ECT and one week post completion of ECT. The difference was highly significant statistically. ($F=82.53$, $p<0.0001$)

Table 15 shows the inter group comparison between different phases on ACE-R scale. Post Hoc test using Fisher test after ANOVA testing revealed that there was mild significant difference between baseline and first ECT. ($p=0.023$) and the difference between first ECT and 1week post ECT was highly significant statistically ($p<0.0001$) and the difference between baseline and 1 week post ECT was highly significant ($p<0.0001$).

Table-16

Table showing the Mean, Standard Deviation and Repeated Measures ANOVA values in Mania Patients

Test	Mean			S.D			Statistics	
	Before E.C.T	After 1st E.C.T	One week post last E.C.T	Before E.C.T	After 1st E.C.T	One week post last E.C.T	F	P
Digit Symbol Substitution Test	608.12	645.5	555.75	105.28	123.96	99.98	13.49	0.0005
COWA Test	7.5	7.375	8.25	1.77	2.199	2.1213	5.68	0.0156
Color Trial Test 1	178.12	206.5	136.75	71.08	89.14	59.30	12.61	0.0007
Color Trial Test 2	377.85	416.5	326.38	106.48	131.64	115.01	14.9	0.0003
Color Trial Test Total	556	623	463.12	177.09	220.39	172.36	15.42	0.0002
PGI Memory Scale	57.625	54	56.75	4.66	4.38	5.52	3.95	0.0436
ACE-R Test	72.25	68.62	78.12	9.13	7.67	8.30	31.81	<0.0001

Table-17

Table showing the Inter-Group Comparison using Fisher's Post-Hoc Test in Mania Patients

Test	Comparison of groups	Statistics
Digit Symbol Substitution Test	Before ECT and After 1st ECT	0.045
	After 1st ECT and One week post last ECT	0.004
	Before ECT and One week post last ECT	0.011
COWA Test	Before ECT and After 1st ECT	0.685
	After 1st ECT and One week post last ECT	0.021
	Before ECT and One week post last ECT	0.02
Color Trial Test 1	Before ECT and After 1st ECT	0.06
	After 1st ECT and One week post last ECT	0.002
	Before ECT and One week post last ECT	0.018
Color Trial Test 2	Before ECT and After 1st ECT	0.041
	After 1st ECT and One week post last ECT	0.001
	Before ECT and One week post last ECT	0.021
Color Trial Test Total	Before ECT and After 1st ECT	0.045
	After 1st ECT and One week post last ECT	0.001
	Before ECT and One week post last ECT	0.016
PGI Memory Scale	Before ECT and After 1st ECT	0.014
	After 1st ECT and One week post last ECT	0.106
	Before ECT and One week post last ECT	0.554
ACE-R Test	Before ECT and After 1st ECT	0.027
	After 1st ECT and One week post last ECT	<0.0001
	Before ECT and One week post last ECT	0.001

Table-18

Table showing the Mean, Standard Deviation and Repeated Measures ANOVA values in Schizophrenia Patients

Test	Mean			S.D			Statistics	
	Before E.C.T	After 1st E.C.T	One week post last E.C.T	Before E.C.T	After 1st E.C.T	One week post last E.C.T	F	P
Digit Symbol Substitution Test	661.12	667.68	621.62	93.73	100.79	88.54	24.26	<0.0001
COWA Test	6.18	5.88	6.68	5.76	2.65	2.095	3.67	0.0375
Color Trial Test 1	233.56	241.62	212.12	64.28	69.86	60.22	18.41	<0.0001
Color Trial Test 2	447.06	460.31	410.94	95.48	103.18	93.395	16.16	<0.0001
Color Trial Test Total	680.62	701.94	623.06	158.96	172.14	151.89	18.11	<0.0001
PGI Memory Scale	55.93	54.06	53.25	4.040	4.007	3.87	7.82	0.0018
ACE-R Test	64.13	63.68	69.56	10.07	8.60	8.66	42.66	<0.0001

Table-19

Table showing the Inter-Group Comparison using Fisher's Post-Hoc Test in Schizophrenia Patients

Test	Comparison of groups	Statistics
Digit Symbol Substitution Test	Before ECT and After 1st ECT	0.313
	After 1st ECT and One week post last ECT	<0.0001
	Before ECT and One week post last ECT	<0.0001
COWA Test	Before ECT and After 1st ECT	0.289
	After 1st ECT and One week post last ECT	0.007
	Before ECT and One week post last ECT	0.178
Color Trial Test 1	Before ECT and After 1st ECT	0.159
	After 1st ECT and One week post last ECT	<0.0001
	Before ECT and One week post last ECT	<0.0001
Color Trial Test 2	Before ECT and After 1st ECT	0.155
	After 1st ECT and One week post last ECT	0.0002
	Before ECT and One week post last ECT	0.0002
Color Trial Test Total	Before ECT and After 1st ECT	0.154
	After 1st ECT and One week post last ECT	<0.0001
	Before ECT and One week post last ECT	<0.0001
PGI Memory Scale	Before ECT and After 1st ECT	0.007
	After 1st ECT and One week post last ECT	0.277
	Before ECT and One week post last ECT	0.003
ACE-R Test	Before ECT and After 1st ECT	0.535
	After 1st ECT and One week post last ECT	<0.0001
	Before ECT and One week post last ECT	<0.0001

Table-20

Table showing the Mean, Standard Deviation and Repeated Measures ANOVA values in Depression Patients

Test	Mean			S.D			Statistics	
	Before E.C.T	After 1st E.C.T	One week post last E.C.T	Before E.C.T	After 1st E.C.T	One week post last E.C.T	F	P
Digit Symbol Substitution Test	565.25	582.42	533.92	82.00	93.48	80.38	15.61	<.0001
COWA Test	7.58	7.08	8.33	2.64	2.43	2.42	10.63	0.00059
Color Trial Test 1	157.50	170.25	129.83	55.56	66.81	45.25	18.24	<.0001
Color Trial Test 2	342.17	372.83	303.08	92.71	117.31	91.27	12.56	0.00023
Color Trial Test Total	499.67	543.08	432.92	147.82	183.68	135.93	15.03	<.0001
PGI Memory Scale	68.67	66.92	65.83	4.05	4.32	4.00	8.95	0.00143
ACE-R Test	78.17	77.58	82.08	7.07	6.92	5.95	25.39	<.0001

Table-21

Table showing the Inter-Group Comparison using Fisher's Post-Hoc Test in Depression Patients

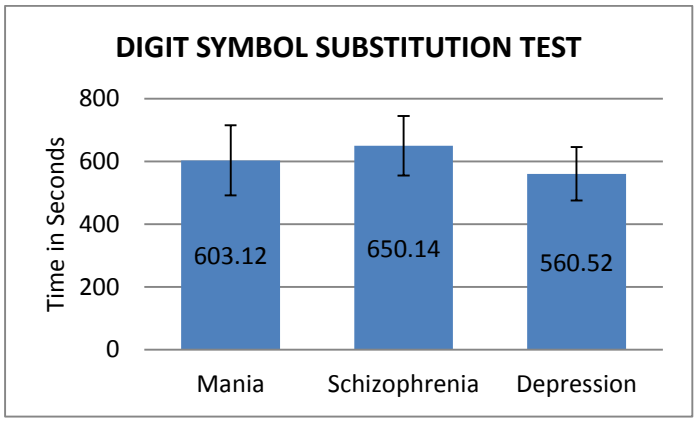
Test	Comparison of groups	Statistics
Digit Symbol Substitution Test	Before ECT and After 1st ECT	0.047
	After 1st ECT and One week post last ECT	0.001
	Before ECT and One week post last ECT	0.002
COWA Test	Before ECT and After 1st ECT	0.082
	After 1st ECT and One week post last ECT	0.003
	Before ECT and One week post last ECT	0.005
Color Trial Test 1	Before ECT and After 1st ECT	0.043
	After 1st ECT and One week post last ECT	0.0005
	Before ECT and One week post last ECT	0.001
Color Trial Test 2	Before ECT and After 1st ECT	0.041
	After 1st ECT and One week post last ECT	0.0006
	Before ECT and One week post last ECT	0.015
Color Trial Test Total	Before ECT and After 1st ECT	0.041
	After 1st ECT and One week post last ECT	0.0004
	Before ECT and One week post last ECT	0.005
PGI Memory Scale	Before ECT and After 1st ECT	0.029
	After 1st ECT and One week post last ECT	0.121
	Before ECT and One week post last ECT	0.002
ACE-R Test	Before ECT and After 1st ECT	0.349
	After 1st ECT and One week post last ECT	<0.0001
	Before ECT and One week post last ECT	0.001

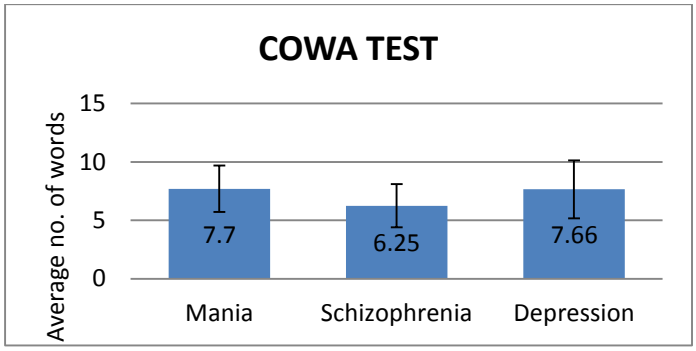
Table-22

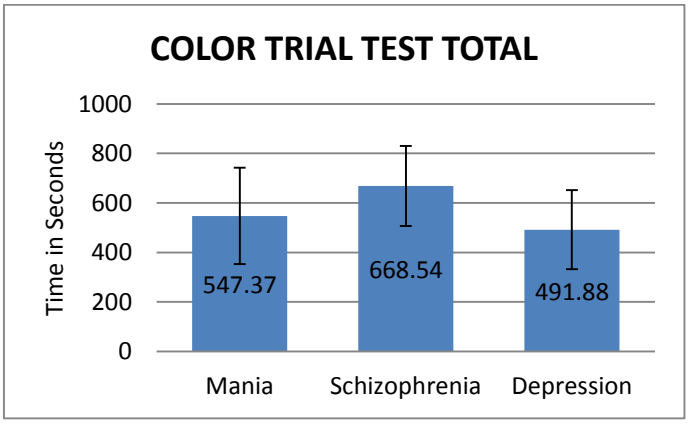
Table showing the Mean difference between before ECT and 1 week post ECT and corresponding Standard Deviation ,t-Test values in Male and Female Patients

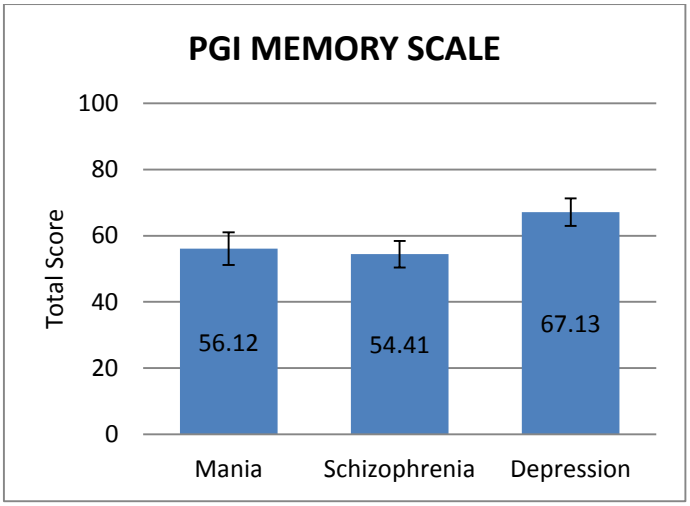
Test	Male		Female		P value
	Mean difference	S.D	Mean difference	S.D	
Digit Symbol Substitution Test	40.39	29.09	38.3	35.1	0.849
COWA Test	-0.39	1.15	-1.07	0.75	0.065
Color Trial Test Total	74.52	58.7	57.76	67.53	0.441
PGI Memory Scale	2.21	2.96	2.53	3.4	0.769
ACE-R Test	-5.26	2.83	-4.61	4.01	0.576

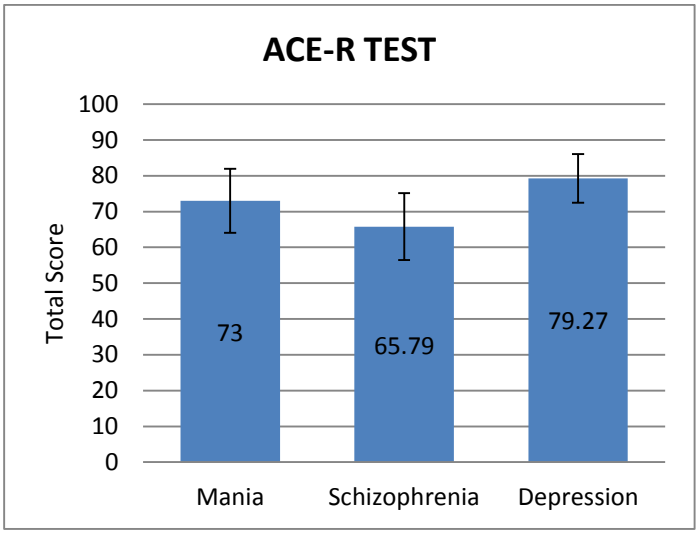
Graphs showing the Mean and Standard Deviation Comparison of Mania, Schizophrenia and Depression Patients in Various Tests








PGI MEMORY SCALE
Total Score
100
80
60
40
20
0
56.12
54.41
67.13
Mania
Schizophrenia
Depression


ACE-R TEST
Total Score
100
90
80
70
60
50
40
30
20
10
0
73
65.79
79.27
Mania
Schizophrenia
Depression

DISCUSSION

The present study attempted to identify the short-term effect of bilateral, brief pulse ECT on cognitive functions among patients who were listed for such treatment in this center. Uniform application of ECT using the same calibrated machine and the same protocols (pulse-width, frequency, time and peak current, bi temporal application of ECT and similar general anesthesia with muscle relaxants) ensured that the delivery of the current was same to all patients. In the present study, the study population was nearly homogenous and the same patient, subsequent data were used to compare the effect of the ECT.

In Digit symbol substitution test which is a sub test of Weschler Intelligence Scale mainly assess the mental speed, sustained attention, visuomotor coordination and motor persistence. In our study population there is a decreasing trend seen immediately after application of first ECT from baseline. Time taken to complete the task increased from baseline score (M-617.39-634.33) which was significant ($p=0.003$). This suggests that ECT has acute effects on attention, mental speed, visuomotor and information processing in the immediate period within 24 hrs of application of ECT. But one week after completion of ECT course revealed that the time taken to complete the task decreased from baseline with a mean score of (M-577.75) and the difference was very much significant ($p<0.0001$) with most of study population performing better when compared to baseline. Thus in our study group it revealed that after completion of ECT course attention, mental speed and information processing were improved and it is revealed by Digit Symbol

substitution test. These attention deficits noted in the acute phase after ECT is consistent with reports of earlier studies.[68, 76, 112]

The amnesic effect of ECT treatment still has not been deciphered completely till date.[55]Many theories have been proposed and several methods have been attempted to reduce these amnesic effects of ECT treatment.[61]Unless the effects of the various domains and characters of this amnesic defects are studied, the mechanism by which ECT causes amnesia could not be completely deciphered. Hence several tools dealing with various domains of memory have been traditionally used by several authors.[66, 69, 71, 76]

In assessing the PGI memory scale scores it was observed that immediately after application of first ECT there was a reduction of mean scores from baseline (M-60.56-58.33). This decline on memory function was significant ($p<0.0001$). On examining the mean score at one week post last ECT it further deteriorated. (M-58.22). This decrease in memory function was not significant when compared to first ECT ($p< 0.842$) but the decline was very much significant when compared to baseline($p<0.0001$). Thus it can be clearly seen that ECT has an adverse cognitive outcome with regards to memory which can be evident even after application of first ECT. Thus it can be observed that with passage of time there is decline in memory and it did not attain baseline or improve after a course of ECT. These findings are consistent with earlier studies and reports.[66,67,69,117] Our study did not take into account the long term impact of ECT on memory in which various studies have different views.ECT affects both retrograde and anterograde memory and in retrograde memory

specifically autobiographical memory is found o be affected in long term.[117] There are studies which have investigated effects of ECT on several intellectual tasks after recovery of orientation after ECT.[23, 25] In such studies authors have used memorized word lists, geometric shapes, nonsense shapes, and neutral and emotional faces before the treatment. After ECT exposure the patients were tested again on recall and recognition immediately after recovery of orientation. Demonstration of memory deficits at this time may in future develop a more long-lasting amnesia. This could be due to secondary to a common and general post-ictal delirium, which is reported in longer term cognitive impairment.

In Controlled Oral Word Association test which is a part of Halstead-Reitan neuropsychological test battery assess the verbal fluency which is a part of executive function there is decline from baseline to first ECT (M-6.94-6.61) in our study group but the decline was insignificant statistically (p=0.05). This effect of ECT on verbal fluency and executive functions has been reported in earlier studies.[73,85] There was significant improvement after completion of course of ECT in the one week period with mean scores improving more than baseline scores (M-6.94-7.58) which is significant statistically (p=0.001). Thus effects seen after first ECT were not seen after completion of course of ECT and resulted in improvement in verbal fluency test.[76]

In Color trial test total mean scores, the time taken to complete the test increased from baseline mean scores (M-592.61- 631.44) which suggests that the patients have declined in their performance on focused attention, perceptual

tracking, simple sequencing and mental flexibility which are measured by these tests after application of first ECT in the immediate period (within 24 hrs of application of ECT). But there is significant improvement in due course which is evident in the post ECT period in which patients completed the task earlier as compared to baseline (M-524.14) which is significant as compared to baseline ($p<0.0001$). Similar findings have been observed in part 1 and part 2 of color trial test. Thus ECT has acute effects on attention in the immediate aftermath of ECT application.[81] ECT also has effects on visuospatial abilities and hemispheric neglect in the acute stages noted immediately after ECT application as seen in earlier studies.[109]

In ACE-R scale scores which measures the overall cognitive performance, it was observed that in the immediate period after application of first ECT, there was decline in cognitive performance in our study population from baseline scores (M-70.61-69.42) but the decline was mildly significant statistically ($p=0.023$). Thus ECT has acute effects on cognitive functions in the immediate period as reported in earlier studies.[86] But after completion of ECT course in the one week post ECT period it was observed that scores improved from baseline (M-75.64) which was highly significant statistically ($p<0.0001$). Thus as passage of time, ECT resulted in improvement in cognitive scores which has been described in several studies. In select individuals, with general anesthesia, post-ictal delirium and or confusion, the MMSE scores have been reported to decrease owing to organic changes. But such a change needs to be at least 25% less of the baseline MMSE scores to be classified as an

organic damage related one. In the present study, none of the patients exhibited a rapid decrease of 25% of the ACE-R scores (which assess the overall cognitive performance like MMSE with extensive scoring) from baseline parameters. This indicates that none of the patients in this study population experienced an organic damage. This findings have been reported earlier by Calev et al.,[76] The transient effects produced by ECT in the acute stages immediately as reported in studies[81-83,114] was found to be resolved in later stages and it ultimately resulted in cognitive improvement by decreasing illness severity . Thus the detrimental effect of ECT is short lasting and after the course of ECT cognitive domain improves over time. These results are very much consistent with earlier studies.[70,76,110,111,115]

On comparing the illness variable it was observed that in digit symbol substitution test schizophrenia patients had taken more time to complete the task (M-661.12) followed by mania patients (M-608.12) and depression group (565.25). In the immediate period post first ECT there was significant decline in scores in mania and depression patients ($p=0.045$ and 0.047) but the decline in scores of schizophrenia patients was insignificant statistically ($p=0.313$). But after completion of ECT course all three groups have improved scores than baseline which was statistically significant from baseline scores ($p=0.011$, $p<0.0001$, $p=0.002$) for mania, schizophrenia and depression group respectively. Thus ECT has resulted in improvement in this task on all three groups.

In PGI memory scale scores, there was significant memory impairment after first ECT in all three groups (p=0.014, p=0.007 and p=0.029) for mania, schizophrenia and depression patients respectively but post one week after completion of ECT mania patients had insignificant memory impairment compared to baseline . Their memory scores neared baseline values (p=0.554) but significant memory impairment persisted in schizophrenia (p=0.003) and depression groups (p=0.002) but the changes from first ECT to one week post ECT were insignificant in all three groups. Thus in mania patients memory neared baseline values but other two groups had significant memory impairment post ECT.

In COWA test ECT resulted in insignificant impairment in verbal fluency in all the three groups with insignificant p values in the immediate period after first application of ECT. After completion of course of ECT it was observed that there was improvement in verbal fluency which was significant in mania and depression groups (p=0.02 and p=0.005) and insignificant in schizophrenia population (p=0.178) from baseline values. Thus improvement in verbal fluency is seen in mania and depression populations but improvement seen in schizophrenia was insignificant after completion of course of ECT.

In color trial test total score there was significant decline in performance in mania and depression groups revealed by increased time to finish the task after first ECT (p=0.045 and p=0.041) respectively. Decline seen in schizophrenia group was insignificant (p=0.154) but after completion of course of ECT time taken to finish the task decreased significantly in all the three

populations from baseline and their baseline scores improved (Focused attention, perceptual tracking and mental flexibility) all improved from baseline and also improved when comparing to first ECT.

In ACE-R scale after measuring immediately post first ECT, significant decline in cognitive scores is seen in mania patients (p=0.027) but the decline seen in schizophrenia and depression groups were insignificant (p=0.535 and p=0.349) respectively. Thus cognitive scores declined in manic populations significantly after application of first ECT. But overall cognitive outcome improved significantly in one week post ECT period compared with first ECT and it even fared better than baseline and there was significant cognitive improvement in all three groups compared to baseline. Thus ECT improves the cognitive outcomes in all three groups. ECT related cognitive effects were independent of type of mental illness.[117] To compare the effects of ECT related cognitive outcomes in male and female populations, mean difference in scores of baseline and one week post ECT were taken into account and the mean difference in male and female populations were compared using t-test . It was noted that in digit symbol test the mean difference in male population was 40.39 and female population was 38.3 and the difference between groups were insignificant (p=0.849). Similarly in PGI memory scale the mean difference in male and female population were 2.21 and 2.53 respectively and the two groups did not differ statistically (p=0.769). Similarly the mean difference in COWA test were -0.39 and -1.07 in males and females and revealed insignificant results (p=0.065).

In color trial test, the male and female population had a score of 74.52 and 57.76 respectively and the groups did not differ significantly in this test (p=0.441) and in ACE-R scale the mean difference between baseline and one week post last ECT in males was -5.26 and females was -4.61 and they did not differ in their outcomes (p=0.576) .Thus it is observed that ECT related cognitive outcomes did not differ according to gender and both male and female populations had similar cognitive outcomes which is seen by measuring the mean difference between baseline and post ECT period in both groups. It did not vary according to gender and same results were seen in different tests.

Since literature evidences favoring ECT by itself causing cognitive improvement are few, any improvement in cognitive outcomes can be attributed to ECT causing illness improvement and recovery thereby indirectly improving cognitive functions.

Advantages of this study are the scales and tests used were administered by the single person therefore interrator bias has been eliminated. ECT has been given to all subjects with same calibrated machine following same protocol thereby eliminating any procedural bias. Three different disorders were compared and cognitive evaluation was made before and after ECT.

The limitation of this study is that it has used only a small sample size with minimal varying factors. The educational and occupational skills have not been taken as potential factors that could cloud the results. In addition, the duration of the disease, stage of the diseases, other co-morbidity (if any) had not been

looked into. The qualitative complaint of the patients or caregiver's feedback has not been accounted. The effect of diminishing memory on quality of life could have been studied, which would shed light in to the deviation from normal life.

CONCLUSION

The current prospective study has evaluated the cognitive outcomes of electroconvulsive therapy. It can be concluded from this study that

- ECT treatment has effects on memory as well as other non memory cognitive functions.
- ECT has acute effects on cognitive functions which are clearly evident by changes in cognitive profile of participating patients seen immediately after first ECT.
- The cognitive effects caused by ECT is short-lived and reversible which is evident by changes in cognitive outcomes from first ECT to one week post ECT period
- ECT ultimately resulted in improved cognitive outcomes on all domains except memory which is evident at one week post ECT period in which all domains showed improved or attained baseline values except memory.
- Furthermore long term effects of ECT on memory has not been taken into account which may have different outcomes on memory
- Gender difference did not play a significant role on ECT related cognitive outcomes.
- Schizophrenia has the most and depression has the least illness related cognitive impairment which is evident by baseline cognitive values of these three disorders.

- ECT resulted in improved cognitive outcomes of all these three disorders except memory. This cognitive improvement seen in these disorders can be attributed to decrease in illness severity by administration of ECT thereby improving cognitive outcomes.

Further studies should include larger sample size with wider tools and having a longer feedback along with biological markers of the brain changes. Such a study would help to decipher the changes that ECT brings on cognitive functions and thereby the quality of the life of these patients.

BIBLIOGRAPHY

1. Fink M, Taylor MA. Electroconvulsive therapy: Evidence and challenges. JAMA 2007;298: 330-35.

2. Franzcp MV. The cognitive side effects of Modern ECT: Patient Experience or Objective Measurements. J ECT 2008; 24:18-24.

3. Freckelton I, Wilson B. Electroconvulsive therapy: Law, history and practice. Journal of Law and Medicine 2001; 8:389-426.

4. Fink M. Meduna and the origins of convulsive therapy. Am J Psychiatry 1984:141:1034-41

5. Taylor S. Electroconvulsive therapy: A review of history, patient selection, technique, and medication management. Southern Medical Journal 2007; 100:494-498.

6. National Institute for Clinical Excellence (2003). Guidance on the use of electroconvulsive therapy: Technology appraisal 59. London: National Institute for Clinical Excellence

7. Fink M. Electroconvulsive Therapy Resurrected: Its Successes and Promises After 75 Years. Can J Psychiatry 2011; 56:3–4.

8. Miller E. Psychological theories of ECT: A Review. Br J Psychiatry, 1967; 113: 301-311.

9. Summerskill J, Seeman W, Meals DW. An evaluation of post electroshock confusion with the Reiter apparatus. American J Psychiatry, 1952; 108: 835-838.

10. Coffey CE, Figiel GS, Djang WT, Cress M, Saunders WB, Weiner RD. Subcortical White matter hyper intensity on magnetic resonance imaging: clinical and neuro anatomical correlates in depressed elderly. J Neuropsychiatry, 1989; 1: 135-144.

11. Pande AC,Grunhaus L, Aisen AM,Haskett RF. A preliminary MRI study of ECT-treated depressed patients. Biol Psychiatry, 1990; 27:102-104.

12. Devanand DP, Shapira B, Petty F, Kramer G, Fitzsimons L, Lerer B, Sackeim HA. Effects of electroconvulsive therapy on plasma GABA. Convulsive Ther, 1995; 11: 3-13.

13. Lawson JS, Inglis J, Delva NJ, et al. Electrode placement in ECT: cognitive effects. Psychol Med, 1990; 20: 335- 344.

14. Charlton BG. The antidelirium theory of electroconvulsive therapy action. Medical Hypothesis, 1999; 52: 609- 611.

15. Weiner RD. Does ECT cause brain damage? Behave Brain Sci, 1984; 7:1-53.

16. Eriksson PS, Perfileva E, Bjork-Eriksson T. Neurogenesis in the adult human hippocampus. Nat Med, 1998; 4: 1313-1317.

17. Gloud E, Beylin A, Tanapat P. Learning enhances adult neurogenesis in the hippocampal formation. Nat. Neurosci, 1998; 2: 260-265.

18. Shah PJ, Ebmeier KP, Glabus MF, Goodwin GM. Cortical grey matter reductions associated with treatment – resistant chronic unipolar depression. Controlled magnetic resonance imaging study. Br J Psychiatry, 1998; 172: 527-532.

19. Sheline YI, Sanghav M, Mintun MA, Gado MH. Depression duration but not age predicts hippocampal volume loss in medically healthy women with recurrent major depression. J Neurosci, 1999; 19: 5034-5043.

20. Kalinowsky L.B, Kennedy F. Observation in electroschock therapy applied to problems in epilepsy. J Nerve Ment Dis, 1943; 98:56-67.

21. Issac L, Schoenbeck R, Bacher J, Skolnick P, Paul SM. Electroconvulsive shock increases endogenous monoamine oxidase inhibitor activity in brain and cerebrospinal fluid. Neurosci Lett, 1986; 66:252-262.

22. Holaday JW, Tortella FC, Meyerhoff JL, Belenky GL, Hitzemann RJ. Electroconvulsive shock activates endogenous opioid systems: behavioral and biochemical correlates. Ann N Y Acad Sci, 1986; 462:249-255.

23. Sackeim HA, Decina P, Prohovnik I. Portnoy S, Kanzler M, Malitz S. Dosage, seizure threshold and the antidepressant efficacy of electroconvulsive therapy. Ann N Y Acad Sci, 1986; 462:398-410.

24. Sackeim HA, Decina P,Portnoy S,Neeley P, Malitz S. Studies of dosage seizure threshold, and seizure duration in ECT. Biol Psychiatry, 1987c; 22: 249-268.

25. Nobler MS, Sackeim HA, Solomou M, Luber B, Devanand DP, Prudic J. EEG manifestations during ECT: effects of electrode placement and stimulus intensity. Biol Psychiatry, 1993; 34:321-330

26. Volkow ND, Bellar S, Mullani N, Jould L, Dewey S. Effects of electroconvulsive therapy on brain glucose metabolism: a preliminary study. Convulsive Ther, 1988.

27. Bonne O, Krausz Y, Shapira B, Bocher M, Karger H, Gorfine M, Chistin R, Lerer B. Increased cerebral blood flow in depressed patients responding to electroconvulsive therapy. J Nucl Med, 1996; 37: 1075-1080.

28. Sackeim HA, Luber B, Katzman GP, Moeller JR, Prudic J. Devanand DP, Nobler MS. The effects of electroconvulsive therapy on quantitative electroencephalogram: relationship to clinical outcome. Arch Gen Psychiatry, 1996; 53:814-824.

29. Abrams R, Volavka J, Schrift M. Brief pulse ECT in melancholia: EEG and clinical effects. J Nerv Ment Dis, 1992; 180: 55-57.

30. Leuchter AF, Cook IA, Uijdehaage SH, Dunkin J, Lufkin RB, Anderson-Hanley C, Abrams M, RosenbergThompson S,O'Hara R, Simon SL, Osato S, Babaie A. Brain structure and function and the outcomes of treatment for depression. J Clin Psychiatry, 1997; 58 (Suppl 16): 22-31.

31. Slade AP & Checkley SA. A neuroendocrine study of the mechanism of action of ECT. Br J Psychiatry , 1980; 137: 217-221.

32. Mann JJ, Mahler JC, Wilner PJ, et al. Normalisation of blunted lymphocyte ß-adrenergic responsivity in melancholic inpatients by a course of electroconvulsive therapy. Arch Gen Psychiatry, 1990; 47: 461-464.

33. Cooper SJ, Kelley JG, King DJ. Adrenergic receptors in depression. Effects of electroconvulsive therapy. Br J Psychiatry, 1985; 147: 23-29.

34. Smith CB, Hollingworth PJ, Garcia-Sevilla JA, Zis AP. Platelet alpha2 adrenoceptors are decreased in number after antidepressant therapy. Prog. Neuropsychopharmacol Biol Psychiatry, 1983; 7: 241-247.

35. Vetulani J, Antkiewicz-Michaluk L, Rokosz-Pelc A, Pilc A. Chronic electroconvulsive treatment enhances the density of [3H] prazosin binding sites in the central nervous system of the rat. Brain Res, 1983; 275:392- 395.

36. Devanand DP, Shapira B, Petty F, Kramer G, Fitzsimons L, Lerer B, Sackeim HA. Effects of electroconvulsive therapy on plasma GABA. Convulsive Ther, 1995; 11: 3-13.

37. Naylor P, Stewart CA, Wright SR, Pearson RC, Reid IC.Repeated ECS induces GluR1 mRNA but not NMDAR1A-G mRNA in the rat hippocampus. Brain Res Mol Brain Res, 1996; 351-2:349-53.

38. Krahn LE, Gleber E, Rummans TA, Pileggi T S, Lucas DL, Li H. The effect of electroconvulsive therapy on melatonin. J ECT, 2000; 16: 391-398.

39. Abrams R, Taylor MA. Diencephalic stimulation and the effects of ECT in endogenous depression. Br J Psychiatry. 1976; 129: 482-5.

40. Deakin JWF, Ferrier IN, Crow TJ, Johnstone EC, Lawler P. Effects of ECT on pituitary hormone release: relationship to seizure, clinical variables, and outcome. British Journal of Psychiatry, 1983; 46: 331-335.

41. Lisanby SH, Devanand DP, Prudic J, Pierson D, Nobler MS, Fitzsimons L, Sackeim HA. Prolactin response to electroconvulsive therapy: effect of electrode placement and stimulus dosage. Biol Psychiatry, 1998; 43: 146-155.

42. Mathe AA.Neuropeptides and electroconvulsive treatment.J ECT. 1999 Mar; 15(1):60-75.

43. Lindefors N, Brodin E, Metsis M. Spatiotemporal selective effects on brain-derived neurotropic factor and TrKB messenger RNA in rat hippocampus by electroconvulsive shock. Neuroscience, 1995; 65:661-70.

44. Metsis M, Timmusk T, Arenas E, Persson H. Differential usage of multiple brain-derived neurotrophic factor promoters in the rat brain following neuronal activation. Proc Natl Acad Sci USA, 1993; 90:8802-8806.

45. Devanand DP, Dwork AJ, Hutchinson ER, Bolwig TG, Sackeim HA. Does ECT alter brain structure? Am. J. Psychiatry 1994:151;957–97

46. Coffey CE, Weiner RD, Djang WT, Figiel GS, Soady SA, Patterson LJ, Holt PD, Spritzer CE, Wilkinson WE. Brain anatomic effects of electroconvulsive therapy. A prospective magnetic resonance imaging. Study. Arch. Gen. Psychiatry 1991; 48:1013–1021.

47. Gregory-Roberts EM, Naismith SL, Cullen KM. Electroconvulsive therapy-induced persistent retrograde amnesia: could it be minimized by ketamine or other pharmacological approaches? Journal of Affective Disorders 2010;126: 39–45

48. Squire LR, Stark CE, Clark RE. The medial temporal lobe. Annu Rev Neurosci.2004;27:279-306.

49. Cardoso A, Assuncao M, Andrade JP, Pereira PA, Madeira MD, Paula-Barbosa MM, et al., Loss of synapses in the entorhinal-dentate gyrus pathway following repeated induction of electroshock seizures in the rat. J Neurosci Res 2008;86(1):71-83.

50. Madsen TM, Yeh DD, Valentine GW, Duman RS. Electroconvulsive seizure treatment increases cell proliferation in rat frontal cortex. Neuropsychopharmacology 2005;30(1):27-34.

51. Dwork, AJ, Arango V, Underwood M, Ilievski B, Rosoklija G, Sackeim HA, Lisanby SH. Absence of histological lesions in primate models of ECT and magnetic seizure therapy. Am. J. Psychiatry 2004;161:576–578.

52. Jinno S, Kosaka T. Reduction of Iba1-expressing microglial process density in the hippocampus following electroconvulsive shock. Exp Neurol 2008;212(2):440-7.

53. Ekdahl CT, Kokaia Z, Lindvall O. Brain inflammation and adult neurogenesis: the dual role of microglia. Neuroscience 2009;158(3):1021–9.

54. Graeber MB, Streit WJ. Microglia: biology and pathology. Acta Neuropathol 2010;119(1):89-105.

55. Andrade C. Molecular mechanisms underlying electroconvulsive therapy-induced amnestic deficits: A decade of research. Indian J Psychiatry 2008;50:244-52

56. Bliss TV, Collingridge GL. A synaptic model of memory: long-term potentiation in the hippocampus. Nature 1993; 361(6407):31-9.

57. Stewart CA, Reid IC. Ketamine prevents ECS-induced synaptic enhancement in rat hippocampus. Neurosci Lett 1994;178(1):11-4.

58. McDaniel WW, Sahota AK, Vyas BV, Laguerta N, Hategan L, Oswald J. Ketamine appears associated with better word recall than etomidate after a course of 6 electroconvulsive therapies. J ECT 2006;22(2):103-6.

59. Horne RL, Pettinati HM, Menken M, Sugerman AA, Varga E, Wilson GF. Dexamethasone in electroconvulsive therapy: efficacy for depression and post-ECT amnesia. Biol. Psychiatry 1984;19: 13–27.

60. Chamberlin E, Tsai GE. A glutamatergic model of ECT-induced memory dysfunction. Harv. Rev. Psychiatry 1998;5:307–317.

61. Andrade C, Thyagarajan S, Singh NM, Vinod PS, Sanjay Kumar Rao N, Chandra JS.Celecoxib as an in vivo probe of cyclooxygenase-2 mechanisms underlying retrograde amnesia in an animal model of ECT. J Neural Transm.2008;115(7):1063-70.

62. Toro CT, Hallak JE, Dunham JS, Leite JP, Sakamoto AC, Guarnieri R, Fonk V, Deakin JF. The NR1 n-methyl D asparatate subunit and brain derived neurotropic factor in temporal lobe epilepsy hippocampus: a comparison of patients with and without co-existing psychiatric symptoms. Epilepsia 2007;48:2352-56.

63. Gurnett CA, Landt M, Wong M. Analaysis of cerebrospinal fluid glial fibrillary acidic protein after seizures in children. Epilepsis 2003;44:1455-58.

64. Lamers KJB, Vos P, Verbeek MM, Rosmalen F, van Geel WJA, van Engelen BGM. Protein S-100B, neuron-specific enolase (NSE), myelin basic protein (MBP) and glial fibrillary acidic protein (GFAP) in cerebrospinal fluid (CSF) and blood of neurological patients. Brain Res bull 2003;61:261-64.

65. Kato K, Ishiguro Y, Suzuki F, Ito a, semba R. Distribution of nervous system-specific forms of enolase in peripheral tissues. Brain res 1982;237:441-48.

66. Sackeim HA, Prudic J, Fuller R, Keilp J, Lavori PW, Olfson M. The Cognitive Effects of Electroconvulsive Therapy in Community Settings. Neuropsychopharmcology 2007; 32:244–54.

67. MacQueen G., et al. (2007). "The long-term impact of treatment with electroconvulsive therapy on discrete memory systems in patients with bipolar disorder". Journal of Psychiatry and Neuroscience **32** (4): 241–249.PMC 1911194. PMID 17653292.

68. Mackenzie, T.B.,Price, T. R. P., Tucker, G. J. & Culver, C.M. (1985). Early change in cognitive performance accompanying bilateral ECT. Convulsive therapy, 1, 183-189.

69. Squire LR, Slater PC. Electroconvulsive therapy and complaints of memory dysfunction: A prospective three-year follow-up study. British Journal of Psychiatry 1983: 142: 1-8.

70. Weeks,D.,Freeman,C. P.L. & Kendell, R.E.(1980). ECT III: Enduring cognitive deficits. British journal of psychiatry, 137, 26-37.

71. Shulze-Rauschenbach SC, Harms U, Schalaepfer TE, Maier W, Falkai P, Wagner M. Distinctive neurocognitive effects of repetitive transcaranial magnetic stimulation and electroconvulsive therapy in major depression. British Journal of Psychiatry 2005;186:410-16

72. McElhiney M, Moody B, Steif B, Prudic J, Debanand D, Nobler M, et al. Autobiographical memory and mood: Effects of electroconvulsive therapy. Neuropsychology 1995;9: 501-17.

73. Crowley K, Pickle J, Dale R, Fattal O. A critical examination of bifrontal electroconvulsive therapy: Clinical efficacy, cognitive side effects, and directions for future research. Journal of Electroconvulsive Therapy 2008;24:268-71.

74. Eschweiler GW, Vonthein R, Bode R, Huell M, Conca A, Peters O, et al. Clinical efficacy and cognitive side effects of bifrontal versus Right unilateral electroconvulsive therapy (ECT): A short-term randomized controlled trial in pharmaco-resistant major depression. Journal of Affective Disorder 2007; 101: 149-57.

75. Bagadia VN, Shah LP, Pradhan PV, Doshi J, Abhyankar R. Evaluation of cognitive effects of ECT. Indian J Psychiat 1981;23:324-29

76. Calev A, Gaudino EA, Squires NK, Zervas IM, and Fink M. ECT and non-memory cognition: A review. British Journal of Clinical Psychology1995: 34:505-15.

77. Viswanath B, Harihara SN, Nahar A, Phutane VH, Taksal A, Thirthalli J, Gangadhar BN. Battery for ECT Related Cognitive Deficits (B4ECT-ReCoDe): Development and validation. Asian Journal of Psychiatry 2013;6(3):243–248

78. Ayuso-Gutierrez JL, Saiz-Ruiz J. The value of cytidine-5-diphosphate-choline in the prevention of impairment of memory functions after electric convulsive therapy. A double-blind study. Prog Neuropsychopharmacol Biol Psychiatry 1982; 6:243-248.

79. Abrams R. Daily administration of unilateral ECT. Amer J Psychiat 1967;124(3):384-386

80. Pettinati HM, Rosenberg J. Memory self-ratings before and after electroconvulsive therapy: depression- versus ECT Induced. Biological Psychiatry 1984;19(4):539-548.

81. Calev, A., Cohen, R., Tubi, N., Nigal, D.,Shapira, B., Kugelmass, S., and Lerer, B. (1991a). Disorientation and bilateral moderately suprathreshold titrated ECT. Convulsive therapy, 7, 99-110.

82. Squire,L.R.(1984). ECT and memory functioning. In B.Lerer,R.D. Weiner & R.H. Belmaker (Eds) ECT: Basic Mechanisms. Pp. 156-163. Washington, DC : American Psychiatric press.

83. Sackeim, H. A. (1992). The cognitive effects of electroconvulsive therapy. In L. J. Thal, W. H. Moos & E. R. Ganzu (Eds), Cognitive Disorders : Pathophysiology and Treatment, p. 183-228. New York : Arnold Dekker.

84. Tubi, N., Calev, A., Nigal, D., Shapira,B., Fink, M., Pass, H., Jandorff, L & Lerer, B. (1993). Subjective symptoms in depression and during the course of electroconvulsive therapy. Neuropsychiatry, Neuropsychology and Behavioral Neurology, 6. 187 192.

85. Taylor, M. A. & Abrams, R. (1985). Short-term cognitive effects of unilateral and bilateral EC'I'. British Journal of' Psychiatry, 146, 308-311.

86. Shapira B, Tubi N, Lerer B. Balancing speed of response to ECT in major depression and adverse cognitive effects: role of treatment schedule. J ECT 2000;16(2):97-109.

87. Sackeim HA, Prudic J, Devanand DP. A prospective, randomized double-blind comparison of bilateral and right unilateral ECT at different stimulus intensities. Arch of Gen Psychiatry 2000;57:425–34.

88. Lisanby SH, Maddox JH, Prudic J, Devanand DP, Sackeim HA. The effects of electroconvulsive therapy on memory of autobiographical and public events. Arch Gen Psychiatry 2000;57:581-590.

89. Bailine SH, Rifkin A, Kayne E, Selzer JA, Vital-Herne J, Blieka M, et al. (2000):

90. Dubovsky SL, Buzan R, Thomas M, Kassner C, Cullum CM. Nicardipine improves the antidepressant action of ECT but does not improve cognition. J ECT 2001; 17:3-10.

91. Tang WK, Ungvari GS, Leung HCI. Effect of piracetam on ECT-induced cognitive disturbances: A randomized, placebo-controlled, double-blind study. J ECT 2002;18(3):130-137.

92. McCall WV, Reboussin DM, Weiner RD, Sackeim HA. Titrated moderately suprathreshold vs fixed high-dose right unilateral electroconvulsive therapy: acute antidepressant and cognitive effects. Arch gen psychiatry 2000; 57:438-444.

93. Tew JD, Jr., Mulsant BH, Haskett RF, Dolata D, Hixson L, Mann JJ. A randomized comparison of high-charge right unilateral electroconvulsive therapy and bilateral electroconvulsive therapy in older depressed patients who failed to respond to 5 to 8 moderate-charge right unilateral treatments. J Clin Psychiatry 2002;63:1102-1105.

94. McCall WV, et al. Markedly suprathreshold right unilateral ECT versus minimally suprathreshold bilateral ECT: antidepressant and memory effects. The Journal of ECT 2002;18: 126-9.

95. Heikman P, Kalska H, Katila H, Sarna S, Tuunainen A, Kuoppasalmi K. Right unilateral and bifrontal electroconvulsive therapy in the treatment of depression: a preliminary study. J ECT 2002;18(1)26-30.

96. Ranjkesh F, Barekatain M, Akuchakian S. Bifrontal versus right unilateral and bitemporal electroconvulsive therapy in m ajor depressive disorder. J ECT. 2005 Dec;21(4):207-10.

97. Chanpattana W, Chakrabhand MLS, Buppanharun W, Sackeim H. Effects of stimulus intensity on the efficacy of bilateral ECT in schizophrenia: A preliminary study. Biol Psychiatry 2000;48:222-228.

98. Prakash J, Kotwal A, Prabhu H. Therapeutic and prophylactic utility of the memory-enhancing drug donepezil hydrochloride on cognition of patients undergoing electroconvulsive therapy: a randomized controlled trial. J ECT. 2006 Sep;22(3):163-8

99. Sackeim, H.A., et al., Effect of concomitant pharmacotherapy on electroconvulsive therapy outcomes: Short-term efficacy and adverse effects. Archives of General Psychiatry, 2009. 66(7): p. 729-737.

100. Geretsegger C, Nickel M, Judendorfer B, Rochowanski E, Novak E, Aichhorn W. Propofol and methohexital as anesthetic agents for electroconvulsive therapy: A randomized, double-blind comparison of electroconvulsive therapy seizure quality, therapeutic efficacy, and cognitive performance. J ECT 2007;23(4):239-243.

101. Stoppe, A., et al., Fixed high-dose electroconvulsive therapy in the elderly with depression: a double-blind, randomized comparison of efficacy and tolerability between unilateral and bilateral electrode placement. J ECT, 2006. 22(2): p. 92-9.

102. Mohan TSP, Tharyan P, Alexander J, Raveendran NS. Effects of stimulus intensity on the efficacy and safety of twice-weekly, bilateral electroconvulsive therapy (ECT) combined with antipsychotics in acute mania: a randomized controlled trial. Bipolar disorders 2009;11:126-134.

103. Hiremani, R.M., et al., Double-blind randomized controlled study comparing short-term efficacy of bifrontal and bitemporal electroconvulsive therapy in acute mania. Bipolar Disorders, 2008. 10(6): p. 701-707.

104. Barekatain M, Jahangard L, Haghighi M, Ranjkesh F. Bifrontal versus bitemporal electroconvulsive therapy in severe m anic patients. J ECT. 2008 Sep;24(3):199-202.

105. Smith GE, Rasmussen KG, Cullum CM, Felmlee-Devine MD, Petrides G, Rummans TA, Husain MM, Mueller M, Bernstein HJ, Knapp RG, O'Connor MK, Fink M, Sampson S, Bailine SH, Kellner CH for the CORE Investigators. A randomized controlled trial comparing

the memory effects of continuation electroconvulsive therapy versus continuation pharmacotherapy: results from the consortium for research in ECT (CORE) study. J Clin Psychiatry 2010;71(2):185-193.

106. Sienaert P, Vansteelandt K, Demyttenaere K, Peuskens J. Randomized comparison of ultra- brief bifrontal and unilateral electroconvulsive therapy for major depression: cognitive side effects. Journal of Affective Disorders 2010;122:60-67.

107. Warnell, R.L., C.M. Swartz, and A. Thomson, Propofol interruption of ECT seizure to reduce side-effects: A pilot study. Psychiatry Research, 2010. 175(1-2): p. 184-185.

108. Kellner CH, Knapp R, Husain MM, Rasmussen K, Sampson S, Cullum M, McClintock SM, Tobias KG, Martino C, Mueller M, Bailine SH, Fink M, Petrides G. Bifrontal, bitemporal and right unilateral electrode placement in ECT: randomized trial. British Journal of Psychiatry 2010;196:226-234.

109. Sackeim,H.A., Nobler, M. S.,Prudic,J., Devanand, D.P., McElhiney, M., Coleman, E.,Settembino, J., Madatta, V.Y. (1992b). Acute effects of electroconvulsive theray on hemispheric neglect. Neuropsychiatry, Neuropsychology and Behavioral Neurology, 5, 151-160.

110. Verwijk E, Comijs HC, Kok RM, Spaans HP, Stek ML, Scherder EJ. Neurocognitive effects after brief pulse and ultrabrief pulse unilateral electroconvulsive therapy for major depression: a review. J Affect Disord. 2012 Nov;140(3):233-43. Doi: 10.1016/j.jad.2012.02.024. Epub 2012 May 15.

111. Neera ghaziuddin, Donna laughrin, and Bruno giordani. " Cognitive side effects of electroconvulsive therapy in adolescents". Journal of Child and Adolescent Psychopharmacology. Winter 2000, 10(4): 269-276. Doi:10.1089/cap.2000.10.269

112. Rami L, Goti J, Ferrer J, Marcos T, Salamero M, Bernardo M. Cognitive functions after only one ECT session: a controlled study. Psychiatry Res 2008;158:389-394.

113. Ng C, Schweitzer I, Alexopoulos P, Celi E, Wong L, Tuckwell V, Sergejew A, Tiller J. "Efficacy and cognitive effects of right unilateral electroconvulsive therapy."JECT. 2000 Dec; 16(4):370-9.

114. Hamilton M, Stocker MJ, Spencer CM. Post-ECT cognitive defect and elevation of blood pressure. Br J Psychiatry. 1979 Jul;135:77-8.

115. Rao V, Lyketsos CG. The benefits and risks of ECT for patients with primary dementia who also suffer from depression. Int J Geriatr Psychiatry. 2000 Aug;15(8):729-35.

116. Sackeim HA, Prudic J, Nobler MS, Fitzsimons L, Lisanby SH, Payne N, Berman RM, Brakemeier EL, Perera T, Devanand DP. Effects of Pulse Width and Electrode Placement on the Efficacy and Cognitive Effects of Electroconvulsive Therapy. Brain Stimulation. 2008 Apr 1;1(2):71-83.

117. Food and Drug Agency, United States of America, Executive Summary. Available from www.fda.gov/downloads/AdvisoryCommittees/CommitteesMeetingMaterials/MedicalDevices/MedicalDevicesAdvisoryCommittee/NeurologicalDevicesPanel/UCM240933.pdf

118. NICE (National Institute for Health and Clinical Excellence) (2009). Depression in Adults (update). National Clinical Practice Guideline 2009; 90:1-585.

119. Pershad D, Wig NN. A battery of simple tests of memory for use in India. Neurol India. 1976;24:86–93.

120. Wechsler, D. (1997). Administration and scoring manual for the WAIS–III. San Antonio, TX: Psychological Corporation.

121. D'Elia et al., 1994. L.F. D'Elia, P. Satz, C.L. Uchiyama, T. White Color Trails Test (CTT) Psychological Assessment Resources, Inc., USA (1994)

122. Benton AL, Hamsher K. Multilingual Aphasia Examination manual. University of Iowa; Iowa City: 1976.

123. Mathuranath PS, Nestor PJ, Berrios GE, Rakowicz W, Hodges JR. A brief cognitive test battery to differentiate Alzheimer's disease and frontotemporal dementia. Neurology 2000;55:1613-20.

ANNEXURES

SEMI STRUCTURED PROFORMA

1) NAME :

2) SEX :1)MALE 2)FEMALE

3) AGE:

4) EDUCATION:1)UNEDUCATED 2)PRIMARY 3)HIGH SCHOOL 4)SECONDARY 5)GRADUATE

5) OCCUPATION:1)EMPLOYED 2)UNEMPLOYED

6) SOCIO ECONOMIC STATUS :1)LOW 2)MIDDLE 3)HIGH

7) MARITAL STATUS:1)MARRIED 2)DIVORCED 3)WIDOW 4)UNMARRIED

8) RESIDENT : 1)RURAL 2)URBAN

9) DIAGNOSIS:1) SCHIZOPHRENIA 2)MOOD DISORDER

10) DURATION OF CURRENT EPISODE :1)LESS THAN ONE MONTH
2)1-6 MONTHS
3)6-12 MONTHS
4)1-2 YEARS
5)>2YEARS

9) FAMILY HISTORY OF PSYCHIATRIC ILLNESS : 1) YES 2)NO

10) NUMBER OF EPISODES: 1)ONE 2)TWO 3)THREE 4)FOUR)>FOUR

11) IF MOOD DISORDER CURRENT EPISODE : 1)MANIA 2)DEPRESSION

12) PAST HISTORY OF PSYCHIATRIC ILLNESS 1)YES 2)NO

13)PAST HISTORY OF PSYCHIATRIC TREATMENT:

14)PAST HISTORY OF MEDICAL ILLNESS: 1)YES 2)NO

15)PAST HISTORY OF MEDICAL TREATMENT:

16)HISTORY OF ECT TREATMENT WITHIN ONE YEAR: 1)YES 2)NO

17)CURRENT PHYSICAL ILLNESS: 1)YES 2)NO

18)IF YES , NATURE OF PHYSICAL ILLNESS:

MASTER CHART

S.No	Name	Sex/Age	Dom	Edu	Marital	SES	Dia	DSST			COWA			CTT 1			CTT 2			CTT T			PGI			ACE-R		
								I	II	III	I	II	III	I	II	III	I	II	III	I	II	III	I	II	III	I	II	III
1	RAMESH	M/28	U	Sec	Sin	Low	S	784	803	716	3	4	4	321	342	283	594	645	524	915	987	807	51	54	53	54	56	62
2	MUTHU KUMAR	M/25	R	Sec	Sin	Mid	S	554	578	523	8	6	8	171	184	157	365	398	332	536	582	489	60	57	56	77	74	80
3	LAKSHMANAN	M/54	R	Pri	Mar	Low	D	726	759	683	4	4	6	249	282	204	485	542	447	734	824	651	62	59	61	66	67	75
4	SUBRAMANIAN	M/30	R	Gra	Mar	Low	S	614	588	574	7	7	7	205	187	179	401	375	351	606	562	530	57	59	52	66	68	73
5	JEBA RATHNAM	F/33	U	Sec	Mar	Mid	M	513	498	442	8	9	9	117	93	79	287	242	196	404	335	275	63	57	65	82	80	89
6	PARAMESHWARI	F/30	R	Gra	Mar	Low	S	766	785	715	4	5	6	301	328	287	550	587	506	851	915	793	52	52	52	55	58	63
7	PERUMAL	M/40	R	Sec	Mar	Low	S	597	611	546	9	7	8	185	199	165	396	418	353	581	617	518	58	56	56	71	70	77
8	SANTHANA KUMAR	M/22	R	Sec	Sin	Low	D	612	651	577	7	8	8	197	211	158	431	474	376	628	685	534	67	69	67	76	75	80
9	MUTHU KUMAR P	M/23	U	Sec	Sin	High	M	521	597	487	6	6	8	129	153	83	302	365	223	431	518	306	55	50	57	69	67	76
10	KANAGALAKSHMI	F/28	U	Pri	Sin	Mid	S	685	705	617	5	5	7	252	283	230	463	503	429	715	786	659	55	52	47	61	59	63
11	NALLA THAI	F/38	R	Pri	Mar	Low	D	484	469	451	12	10	13	94	84	79	251	233	185	345	317	264	73	68	70	88	84	89
12	RAJAN	M/35	R	Sec	Sin	Mid	S	707	693	673	4	6	6	275	261	254	476	455	433	751	716	687	54	49	49	58	55	63
13	MOBIN	M/24	R	Sec	Sin	Low	M	684	721	604	6	5	6	238	258	156	463	511	385	701	769	541	53	51	52	64	63	70
14	VANITHA	F/31	U	Gra	Mar	Mid	S	746	787	692	4	4	6	290	327	255	495	547	438	785	874	693	52	51	50	54	53	58
15	ARUNA	F/28	R	Sec	Mar	Low	M	632	674	583	9	8	10	195	239	138	417	449	392	612	688	530	60	61	59	76	66	82
16	SHANMUGA AYYA	M/54	R	Pri	Mar	High	D	668	698	633	6	6	6	226	252	184	446	520	380	672	772	564	63	62	61	70	68	73
17	KANI RAJ	M/55	R	Sec	Mar	Low	D	532	533	504	5	5	7	138	126	102	331	296	238	469	422	340	66	64	65	73	74	81
18	MUTHU KUMARAN	M/28	R	Pri	Sin	Mid	S	651	626	628	6	5	5	238	217	226	467	441	416	705	658	642	56	53	53	64	64	68
19	MANIMALA	F/30	U	Sec	Mar	Low	D	552	519	496	7	6	8	163	148	119	360	328	312	523	476	431	71	72	69	81	84	87
20	MAAN RAJ	M/23	R	Sec	Sin	Mid	S	805	783	759	5	5	7	336	325	287	613	584	573	949	909	860	50	49	52	50	55	62
21	ARUMUGASUNDARI	F/37	R	Pri	Mar	Low	M	553	655	512	10	11	11	141	209	109	348	423	304	489	632	413	57	51	58	73	68	82
22	MALLIGA	F/20	R	Sec	Sin	Low	S	593	620	613	8	7	8	193	217	204	415	447	432	608	664	636	59	55	50	73	67	69
23	JOHN	M/23	R	Sec	Sin	Mid	S	538	507	487	10	8	9	157	133	121	341	301	298	498	434	419	61	56	58	78	75	82
24	SIMSON	M/29	R	Gra	Mar	Mid	D	593	629	534	6	5	6	174	204	136	374	441	329	548	645	465	68	65	66	78	78	85
25	ALEX PANDIYAN	M/26	R	Sec	Sin	Low	M	696	737	719	7	6	7	184	262	218	392	480	447	576	742	665	56	50	47	67	62	69
26	RAMALAKSHMI	F/30	U	Sec	Mar	High	S	739	778	679	4	4	6	277	298	255	492	537	445	769	835	700	53	52	52	55	55	64
27	VELLA THAI	F/40	R	Pri	Mar	Low	D	628	658	601	9	10	9	218	245	178	435	494	393	653	739	571	66	63	59	75	71	76
28	MUTHU KUMAR S	M/27	R	Sec	Mar	Mid	M	487	456	448	9	9	10	106	91	85	243	239	187	349	330	272	65	59	61	87	81	88
29	CHANDRA BABU	M/34	R	Sec	Mar	Low	D	508	489	485	8	7	9	104	90	81	236	205	185	340	295	266	72	71	69	85	85	87
30	PALANI KUMAR	M/26	U	Gra	Sin	Mid	S	487	504	456	11	10	9	104	132	88	231	274	196	335	406	284	65	65	63	84	82	88
31	GOMATHI	F/30	R	Pri	Sin	Mid	D	514	544	563	7	6	7	119	143	151	257	322	358	376	465	509	69	71	67	75	76	77
32	KARUPPASAMY	M/27	R	Pri	Mar	Low	S	678	703	691	6	6	5	225	246	232	430	466	484	655	712	716	55	53	53	60	61	66
33	SUBASH RAJ	M/26	U	Sec	Sin	High	M	779	826	651	5	5	5	315	347	226	571	623	477	886	970	703	52	53	55	60	62	69
34	NAAMA SELVAM	M/37	U	Pri	Mar	Low	D	442	491	404	13	12	13	83	106	72	222	258	196	305	364	268	75	72	72	89	88	91
35	SAMUTHIRAVALLI	F/28	R	Pri	Mar	Mid	S	634	612	577	5	5	6	207	187	171	424	387	365	631	574	536	57	52	56	66	67	75
36	MICHAEL RAJA	M/23	R	Sec	Sin	Mid	D	524	549	476	7	6	8	125	152	94	278	361	238	403	513	332	72	67	64	82	81	84

ADDENBROOKE'S COGNITIVE EXAMINATION – ACE-R

Administration and Scoring Guide - 2006

The ACE-R[1] is a brief cognitive test that assesses five cognitive domains, namely attention/orientation, memory, verbal fluency, language and visuospatial abilities. Total score is 100, higher scores indicates better cognitive functioning.
Administration of the ACE-R takes, on average, 15 minutes.
These instructions have been designed in order to make the questions and their scoring clear for the tester. Please read them carefully before giving the test.
If possible, leave the scoring until the end of the session, since the participant will not be able to check whether the tester is ticking for correct answers or crossing for wrong ones. This might avoid anxiety, which can disturb the participant's performance on the test.

ORIENTATION – score 0 to 10

Ask the participant for the day, date, month, year and season. Score one point for each correct answer.

Ask the participant for the name of the hospital (or building), the floor (or room), the town, county and country. Score one point for each correct answer.

Record responses. Allow mistakes for the date (+ or – 2 days). If assessing a participant at home, ask for the name of the place i.e. name of the house e.g. "The Gables", and for the floor you might ask for the name of the room (kitchen, living room, etc). If at a single storey health setting, ask about a local landmark. When the season is changing, e.g. at the end of August, and the participant says "autumn", ask them "could it be another season?". If answer is "summer", give one point, since the two seasons are in transition. Do not give one point if the answer is "winter" or "spring".

Seasons: spring - March, April, May; summer - June, July, August; autumn - September, October, November; winter - December, January, February.

REGISTRATION – score 0 to 3

Ask the participant to repeat and remember the words lemon, key, and ball. Speak slowly. Repeat them if necessary (maximum 3 times). Tell the participant that you will ask for this information later. Record the number of trials. Score the first attempt only.

ATTENTION & CONCENTRATION – score 0 to 5

Calculation: Ask the participant to subtract 7 from 100, record the answer, then ask them to subtract 7 from that, record the answer. Do this 5 times. If the participant makes a mistake, carry on and check subsequent answers for scoring. Record responses (Example: 92, 85, 79, 72, 65, score 3).

Spelling: give this test if the participant makes a mistake on the calculation task. Start by asking the participant to spell "world". Then ask them to spell it backwards. Record responses.

Scoring for the spelling task:
- Score 1 point for each correct letter spelt. Correct sequence = D L R O W = 5 points
- Count one error for each omission, letter transposition (switching adjacent letters), insertion (inserting a new letter), or misplacement (moving W, O, R, L, D by more than one space).

Examples (score in parentheses):

	omission	transposition	insertion	misplacement
omission	DLOW (4)			
transposition	D***OL***W (3)	DL***OR***W (4)		
	omission	transposition	insertion	misplacement
insertion	DL***T***OW (3)	DLR***WWO*** (3)	DL***RR***OW(4)	
misplacement	LOW***D*** (3)	LR***WOD*** (3)	LR***W***OW***D*** (3)	LROW**D** (4)

A response such as 'LRWWOD' has 3 errors (L and R are correct, for a score of 2). It includes transposition of the W and O, insertion of an extra W, and misplacement of the D. If the patient adds 1 or more of the same letter at the end of the word, count as one error (e.g. ' LDROWWW, would be 2 errors, 1 transposition and 1 addition).

Score one point for each correct calculation or letter spelt. Score only the better performed one.

R E C A L L – score 0 to 3

Ask the participant to recall the words that you asked them to repeat and remember.
Record responses. Score one point for each correct item.

Anterograde Memory – score 0 to 7

Instruct the participant: "I'm going to read you a name and address that I'd like you to repeat after me. We'll be doing that 3 times, so you have a chance to learn it. I'll be asking you about it later". If the participant starts repeating along with you, ask them to wait until you give it in full.
Record responses for each trial. However, only the third score contributes to the ACE-R score (0-7points).

Retrograde Memory – score 0 to 4

Ask the participant for the name of the current Prime Minister, the woman who was Prime Minister, the president of the US and the president of the US who was assassinated in the sixties.
Score one point each. Allow answers like Blair, Thatcher; Bush; Kennedy. Do not accept answers like Maggie, ask for surname as well.

V E R B A L F L U E N C Y – score 0 to 14

Letters – score 0 to 7

Instruct the participant: "I'm going to give you a letter of the alphabet and I'd like you to generate as many words as you can beginning with that letter, but not the names of people or places. Are you ready? You've got a minute and the letter is P".
Participant might repeat or perseverate words, e.g. pay, paid, pays. Record and count them for the overall total number of responses but do not consider them for the final score. In the same way, intrusions such as words beginning with other letters are recorded but not scored. Proper names (Peter, Peterborough) do not count. For plurals e.g. pot, pots, total = 2, correct = 1. Use the table provided on the ACE-R sheet to obtain the final score for this test.

Animals – score 0 to 7

Instruct the participant: "Now can you name as many animals as possible, beginning with any letter?"
Participant might repeat words. Record and count them for the overall total number of responses, but they should not be considered for the final score. The participant may misunderstand and perseverate by naming animals beginning with "p". Repeat instructions during the 60 seconds if necessary.
If subject says e.g. "fish", and later says "salmon" and "trout", count and record as 3 for "total" but do not accept "fish" as correct (count only 2 out of the 3, e.g. "salmon" and "trout"). However, if only the category is given, e.g. fish, with no specific exemplars, then count fish as 1 for total and final correct responses. The same applies to mammals, reptiles, birds, breeds of dog, insects, etc.

no specific exemplars, then count fish as 1 for total and final correct responses. The same applies to mammals, reptiles, birds, breeds of dog, insects, etc.

L A N G U A G E - Comprehension (Close your eyes) – score 0 or 1

Instruct the participant: "Read this sentence and do as it says". If the participant reads sentence aloud but does not follow the instructions, score 0.

L A N G U A G E - Comprehension (3-stage command) - score 0 to 3

Instruct the participant: "Take this paper in your right hand, fold it in half, and put it on the floor". Do not allow participant to take the paper before you have finished giving the complete instruction.
Score one point for each correct command, e.g. if participant takes the paper and puts it on the floor without folding, score 2; if participant takes the paper in their right hand, and folds it several times and leaves on the table, score 1.

L A N G U A G E - Writing – score 0 or 1

Instruct the participant to write a sentence.
The sentence should contain a subject and a verb, and it should have a meaning.
Do not accept "Happy Birthday" or "Nice day" as a sentence. If participant has difficulty thinking of something to write, prompt gently with "What's the weather like today?"

L A N G U A G E - Repetition – score 0 to 2

Ask the participant to repeat the words after you. Say one word at a time. Circle the words that were repeated incorrectly. Consider first attempt only for scoring. Record responses. Score 2 if all words are correct; 1 if 3 are correct; 0 if 2 or less are correct.

L A N G U A G E - Repetition – score 0 to 2

Ask the participant to repeat each sentence. Do not accept partially correct repetitions, e.g. "no ifs and buts", "above below" as correct for scoring. Score one point for each sentence.

L A N G U A G E - Naming – score 0 to 2

Naming (watch and pencil)
Ask the participant to name each picture. Correct answers: pencil; wristwatch or watch.

L A N G U A G E - Naming – score 0 to 10

Naming (5 animals and 5 objects)
Ask the participant to name each picture. Correct answers: penguin; anchor; camel or dromedary; barrel or tub; crown; crocodile or alligator; harp; rhinoceros or rhino; kangaroo or wallaby; piano accordion, accordion or squeeze box.
Score one point each.

L A N G U A G E - Comprehension – score 0 to 4

Comprehension
Ask the participant to point to the pictures according to the statement read.
Score one point each. Allow self-corrections.

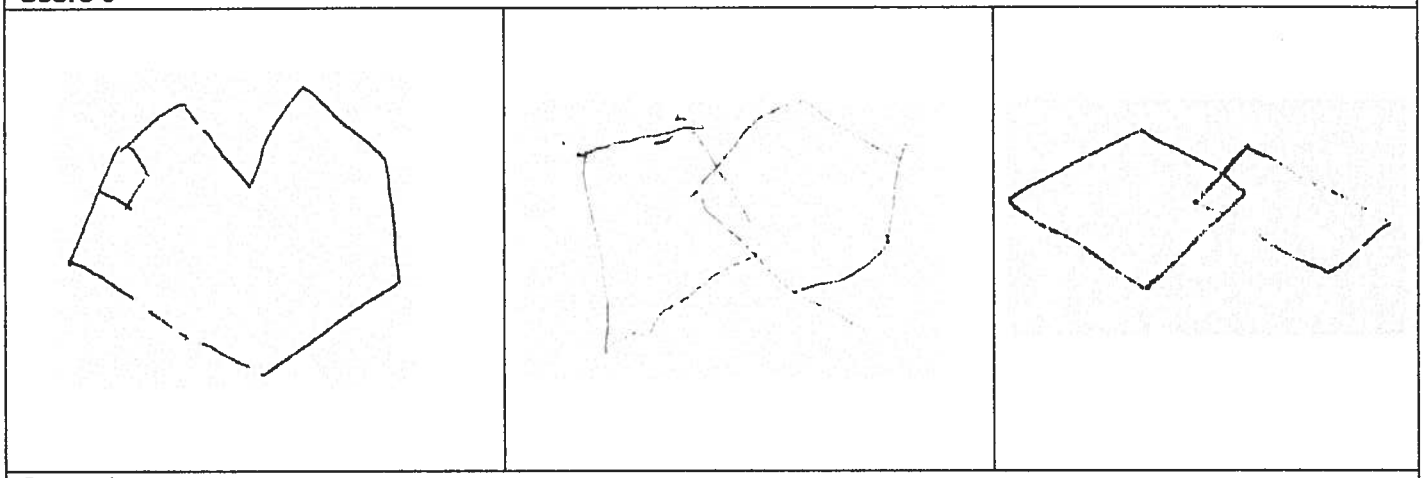
LANGUAGE - Reading – score 0 or 1

Ask the participant to read the words aloud. Score one point only if all five words are correctly read. Record the mistakes using the phonetic alphabet if possible.

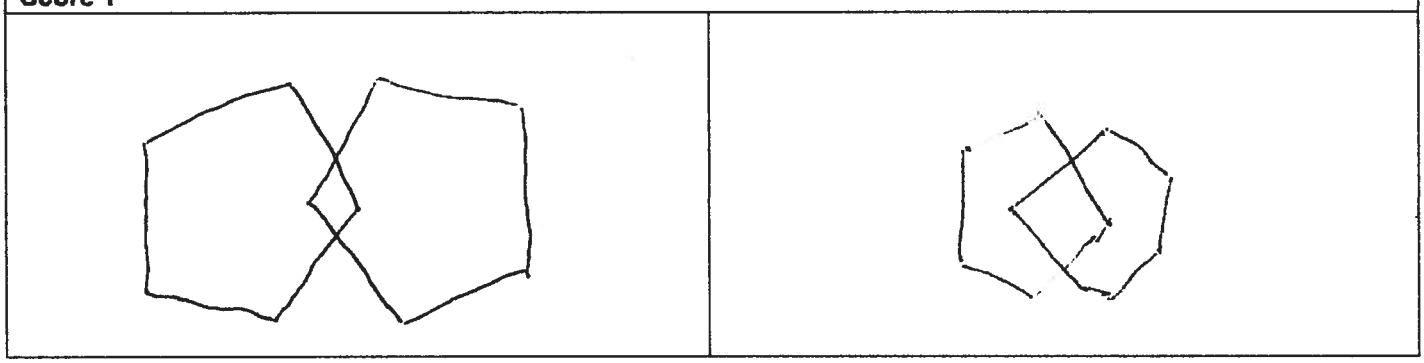
VISUOSPATIAL ABILITIES - Overlapping pentagons – score 0 or 1

The pentagons should clearly show 5 sides and the intersection.

Score 0



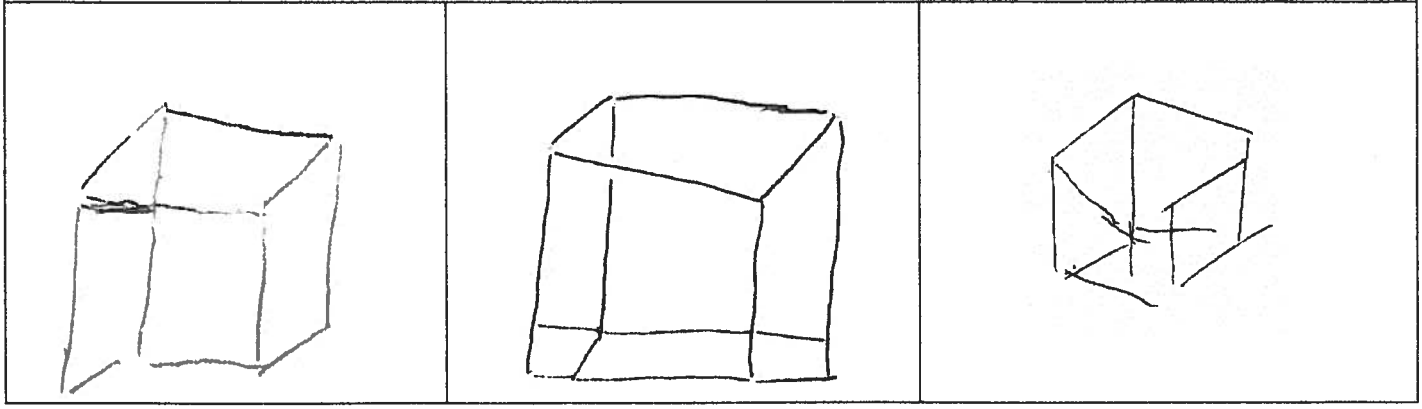
Score 1



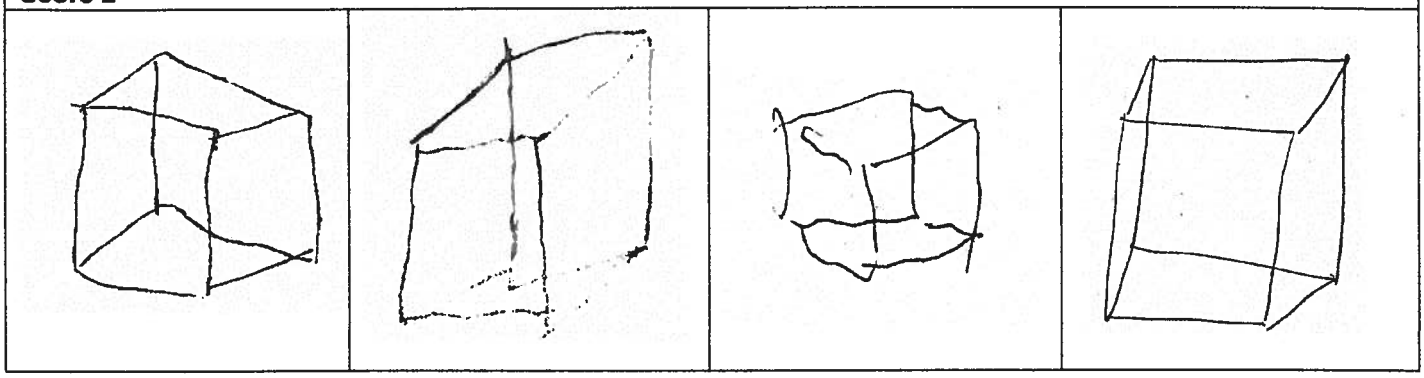
VISUOSPATIAL ABILITIES - Wire Cube – score 0 to 2

Cube should have 12 lines = score 2, even if the proportions are not perfect. A score of 1 is given if cube has fewer than 12 lines, but general cube shape is maintained. See examples below.

Score 1



Score 2



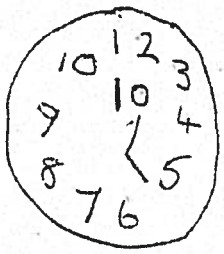
VISUOSPATIAL ABILITIES - Clock – score 0 to 5

Ask the participant to draw a clock face with the numbers on it. When he/she has finished, ask them to put the hands at "ten past five".

Circle	1 point maximum if it is a reasonable circle
Numbers	2 points if all included and well distributed 1 point if all included but poorly distributed
Hands	2 points if both hands are well drawn, different lengths and placed on correct numbers (you might ask which one is the small and big one) 1point if both placed on the correct numbers but wrong lengths OR 1 point if one hand is placed on the correct number and drawn with correct length OR 1 point if only one hand is drawn and placed at the correct number i.e. 5 for 'ten past five'

Score 2

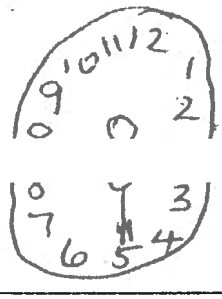
Circle (1); one hand placed correctly (1)



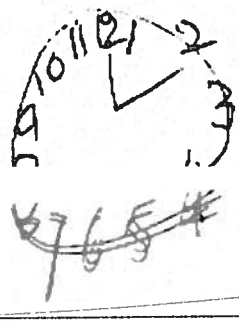
Circle (1); all the numbers but not placed inside the circle (1)

Score 3

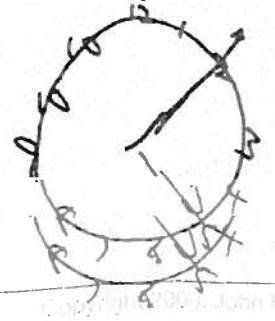
Circle (1); all the numbers but not proportionally distributed (1), one hand placed correctly (1)



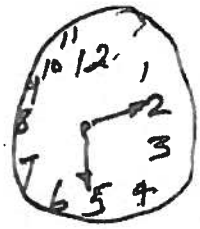
Circle (1), all the numbers but not placed inside the circle (1), one hand place correctly (1).



Circle (1), note that numbers are not inside the circle and there are 2 number 10s (0), hands placed correctly

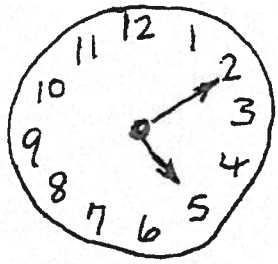


Score 4

Circle(1); numbers proportionally distributed (2); one hand placed correctly (1)	Circle (1); all the numbers but not proportionally distributed (1); both hands placed correctely (2)	Circle (1); numbers proportionally distributed (2), one hand placed correctly (1)
		

Score 5

Circle (1); numbers proportionally distributed on both halves of the clock face (2); hands placed correctly (2)



PERCEPTUAL ABILITIES – score 0 to 4

Counting dots
Participant is *not* allowed to point to the picture. Score one point for each correct answer.
Correct answers, from top left clockwise: 8, 10, 9 and 7.

PERCEPTUAL ABILITIES – score 0 to 4

Identifying letters
Participant is allowed to point to the picture. Score one point for each correct answer.
Correct answers, from top left clockwise: K, M, T and A

RECALL – score 0 to 7

Recall
Say to the participant: "Now tell me what you remember of that name and address we were repeating at the beginning". Tick and score one point for each item recalled, using the score guide provided in the test.

Harry Barnes
73 Orchard Close
Kingsbridge
Devon

Example 1a

Harry Bond 78 Orchard Close Kingsbury	1 + 0 0 + 1 + 1 0 0	**Score 3/7**

Example 2a

Harry Barnes 73 Kingsbridge Close .... Devon	1 + 1 1 + 0 + 1 0 1	**Score 5/7**

Example 3a

Harry Bond 33 Kingsbury Way Kingsbridge Close Cambridge Devon	1 + 0 0 + 0 + 0 0 + 0 0 1	**Score 2/7**

RECOGNITION – score 0 to 5

Recognition – only to be given if participant fails to recall one or more items in the recall task.
This task should be given to allow the participant a chance to recognise items that he or she could not recall. If the participant recalls the name and address correctly, this test is not needed and the participant scores 5. However, many participants will recall only parts. Start by ticking the correctly remembered items on the shadowed column (right hand side) and then tell them "Let me give you some hints. Was the number (or whatever was forgotten or mistaken) x, y or z?" and so on. Every recognised item scores one point. Maximum score is 5. Adding recalled items to those recognised gives the final score for this part of the test.

Example 1b (based on example 1a)

Tester ticks "Orchard Close" on the right hand side shadowed column because participant had recalled that item. The tester should then ask: - Was it Jerry Barnes, Harry Barnes or Harry Bradford? - Was it 37, 73 or 76? - Was it Oakhampton, Kingsbridge or Dartington? - Was it Devon, Dorset or Somerset?	Participant's answers: Harry Barnes 76 Kingsbridge Dorset	 1 0 1 0 + 1 (Orchard Close) **Score 3/5**

Example 2b (based on example 2a)

Tester ticks "Harry Barnes", "73" and "Devon" on the right hand side shadowed column because participant had recalled those items. The tester should then ask: - Was it Orchard Place, Oak Close or Orchard Close? - Was it Oakhampton, Kingsbridge or Dartington?	Participant's answers: Orchard Close Kingsbridge	 1 1 + 3 (Harry Barnes, 73, Devon) **Score 5/5**

Example 3b (based on example 3a)

Tester ticks "Devon", on the right hand side shadowed column because participant had recalled that item. The tester should then ask: - Was it Jerry Barnes, Harry Barnes or Harry Bradford? - Was it 37, 73 or 76? - Was it Orchard Place, Oak Close or Orchard Close? - Was it Oakhampton, Kingsbridge or Dartington?	Participant's answers: Jerry Barnes 37 Orchard Place Oakhampton	 0 0 0 0 +1 (Devon) **Score 1/5**

Digit symbol substitution test

1	2	3	4	5	6	7	8	9
↔	↕	≡	⫼	≠	□	Φ	∈	∋

2	9	2	9	4	9	4	9	1	8	9	3	1	7	2	3	6	4	8	3	1	7	8	2	5

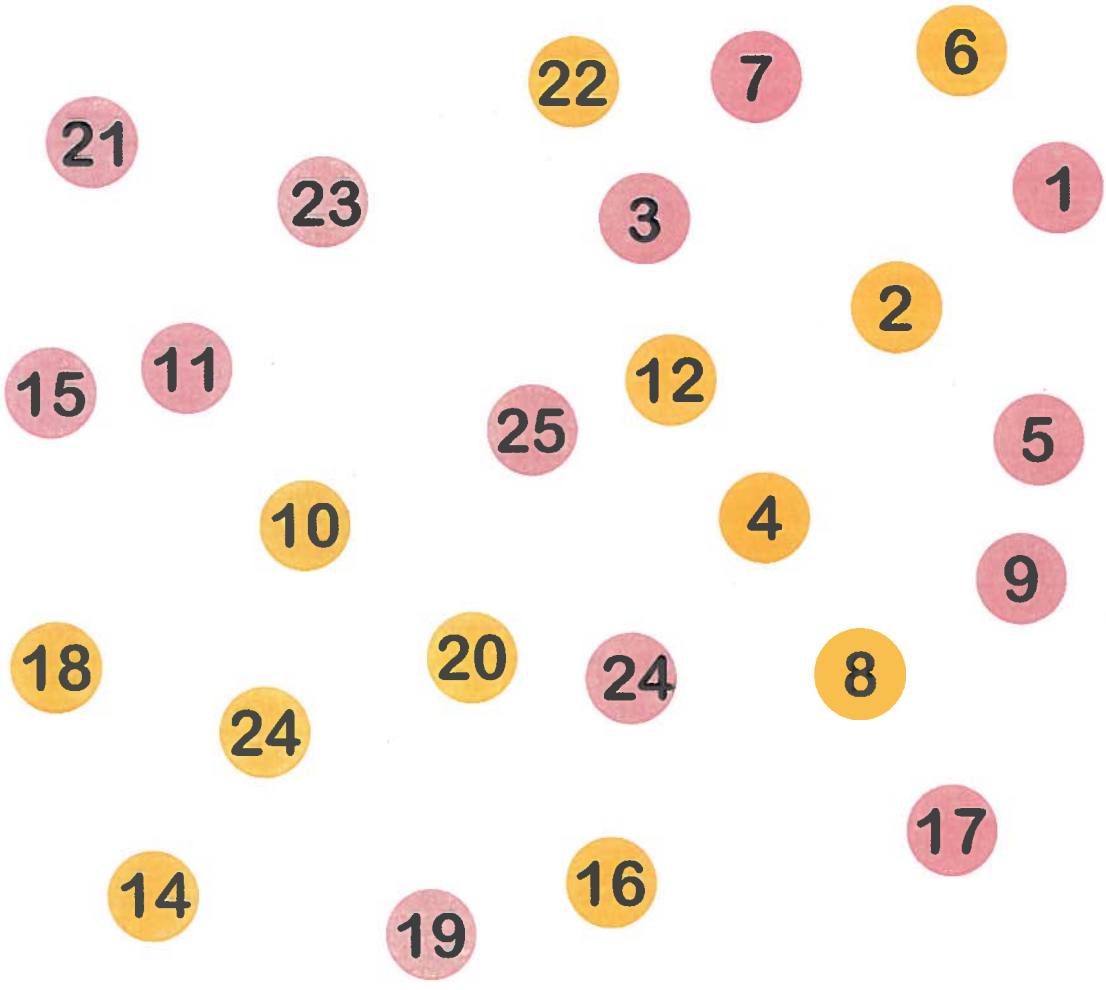
4	7	1	7	5	8	4	1	5	2	6	9	9	5	6	7	6	2	9	4	8	7	2	8	6

8	6	2	8	2	9	4	7	4	8	6	7	3	1	6	2	1	8	7	4	3	1	6	2	9

2	5	4	6	1	6	3	1	2	7	2	6	4	9	1	8	5	7	1	5	4	5	3	9	2

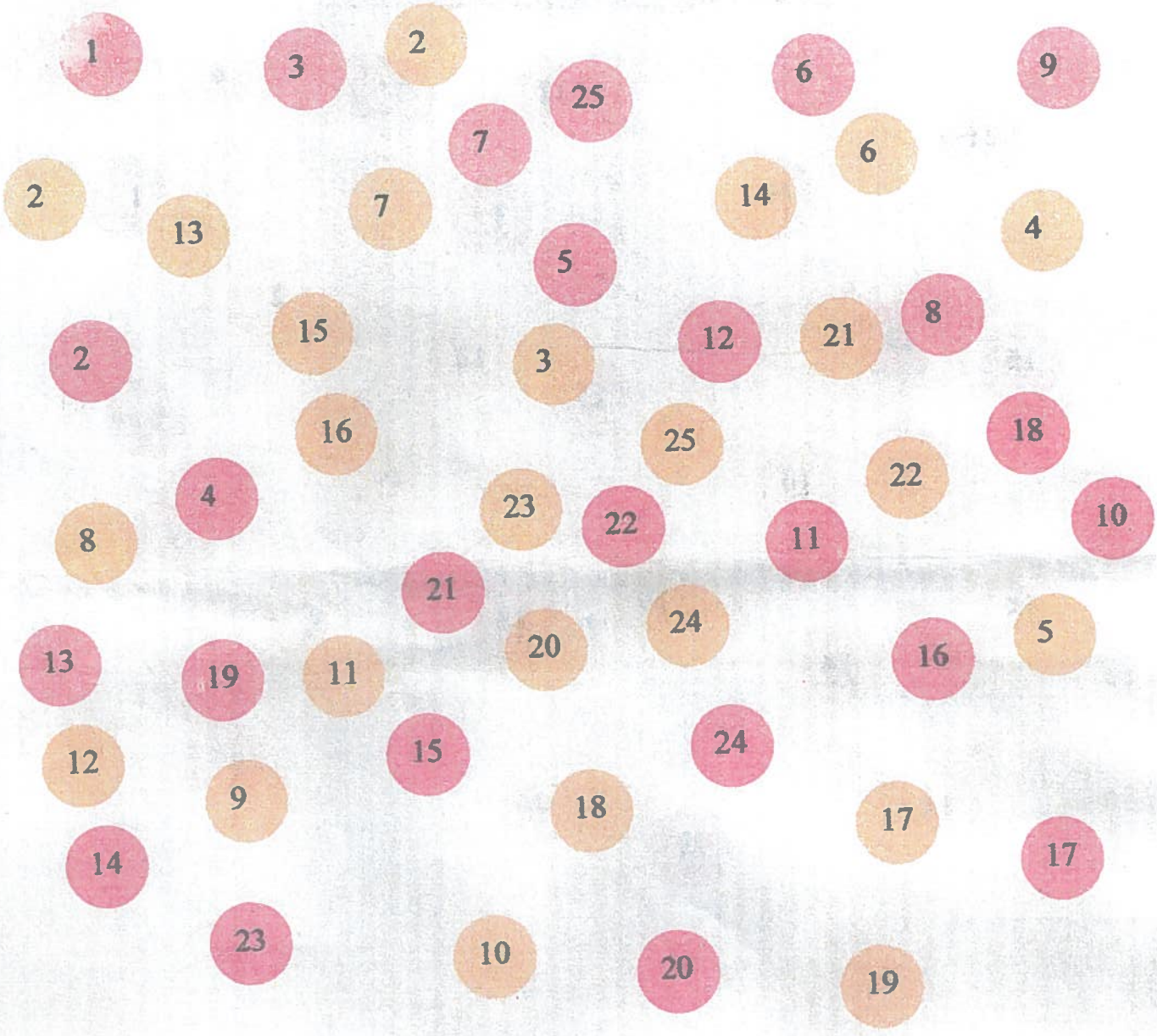
3	9	7	1	7	1	3	5	7	6	1	6	5	9	1	3	1	3	9	8	9	7	3	4	3

COLOR TRAILS TEST SHEET 1



COLOR TRAILS TEST SHEET

2



PGI MEMORY SCALE

I. **Remote Memory**

1. How old are you ? .....
2. Where were you born ? .....
3. (a) When were you married ? .....
   (b) When did you start earning ? .....
   (c) When did you left study/pass high school ? .....
4. How old is your youngest child/brother/sister ? .....
5. When did you come first time in this clinic/department (Hospital) ? .....
6. When did you come here last time ? .....

II. **Recent Memory**

1. What did you eat in your last dinner ? .....
2. What did you eat this morning ? .....
3. What is the name of this month ? .....
4. What day is today ? .....
5. Who came to visit you or to whom you visited yesterday ? .....

III. **Mental Balance**

1. Recite A to Z (Alphabet of any language).
2. Count backward from 20 to 1.
3. Count backward by minusing 3s starting from 40.

IV. **Attention and Concentration**

1. I am going to say some numbers. Listen them carefully, when I reac them, you will repeat them in the same order.

5-7-3	4-1-7
5-3-8-7	6-1-5-8
1-6-4-9-5	2-9-7-6-3
3-4-1-7-9-6	6-1-5-8-3-9
7-2-5-9-4-8-3	4-7-1-5-3-8-6
4-7-2-9-1-6-8-5	9-2-8-8-3-1-7-4

2. I am going to read some numbers but you will be required to repeat them backward. For example, I say 2, 5 you will say 5, 2.

8-5	2-8
4-3-7	8-5-1
8-5-6-3	3-7-5-9
4-7-2-9-1	9-2-5-8-4
2-5-9-4-8-3	7-1-5-3-9-6
3-5-8-6-1-9-2	6-3-7-1-4-8-5
8-5-2-3-6-1-9-4	2-8-4-5-9-7-1-3

V. **Delayed Recall**

I am going to read the name of some objects, listen carefully and when I ask you to repeat, you will do so.

(Read at the rate of one word per second and ask the subject to repeat it after an interval of one minute.)

1	2
Umbrella	Fish
Flower	Lamp
Clock	Rupee
Picture	Taj
Pencil	Toy

Vi. **Immediate Recall**

I am going to read a few small sentences one by one. Listen them carefully because when I am through I would like you to tell me the whole sentence as precisely as you can.

1. Ram got up from the chair, opened the door and went to market.
2. Patient was asked to lie down on the table, he was seen, medicine was prescribed and was told to come next day.
3. Mohan did not have water in his house, he picked-up the bucket, went to street well, filled it up and returned back.

VII. **Verbal Retention for Similar Pairs**

Now, I am going to read to you a list of pairs, i.e., two words at a time. Listen carefully, when I name one word of the pair you will tell the second word of the pair (read the pairs at the rate of 2 seconds per pair, with a pause of 5 seconds between pairs ; give an interval of 10 seconds after reading last pair and then present a stimulus word of the pair and ask the subject to recall the 2nd word of the pair).

1.	Tree	Flower
2.	Sweet	Sour
3.	Man	Woman
4.	Day	Night
5.	Black	White

VIII. **Verbal Retention for Dissimilar Pairs**

Instruction and administration procedure is the same as above for subtest VII, with a difference that stimulus words are to be presented in the order as mentioned for each of the trial. If subject fails to give correct answer, correct it and proceed to next stimulus word. If all his answers are correct in first trial even then other two trials are to be completed but in no case, pairs be repeated, only incorrect answers are to be corrected.

Table	Black	4	2	1
Tree	High	2	1	5
Lamp	Uneven	1	5	3
Child	Bitter	3	4	2
Dream	Deep	5	3	4

IX. Visual Retention

I am going to show you a card, see it carefully. After some time (15 seconds) I will take it away and when I ask you to draw (after 30 seconds) them, draw the things you saw in the card from your memory on this paper (give a paper, a pencil and an erasure to the subject but do not instruct whether he can use the erasure or not).

X. Recognition

I am showing you a card containing pictures of many objects, see the whole card attentively (expose for 30 seconds). After some time (120 seconds interval) I will place before you another card. From this you will be required to identify and name the objects you saw in earlier card (Do not tell the subject, the exact number of objects seen in first card and how many things he is yet to identify).

Scoring Criteria for Various Items of Recognition Sub-test X :—

Give a score of one for correct identification and correct naming of an item.

Give half score for correct identification but wrong naming of an item.

Few subjects sometimes use different names for a particular item. Correct names for each item of this sub-test are given below. Any name other than those given here and only the description of the item, should be regarded as incorrect. However, if the examiner feels that the response given by the subject is not markedly wrong, he may give credit for that.

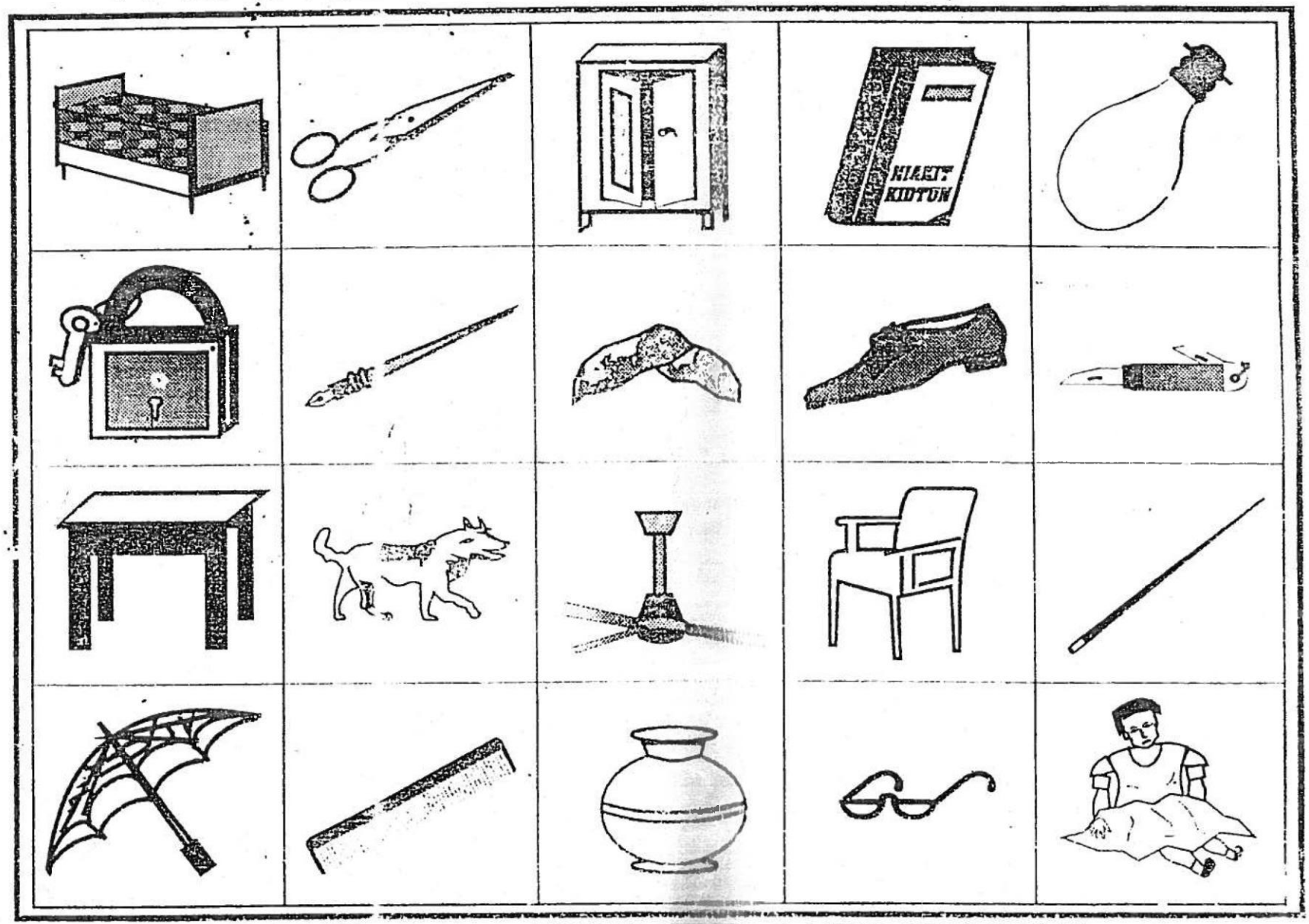
1. Lock = Lock/Lock-key/Close/Sed
2. Pen = Holder/Pencil/Ink
3. Fan = Fan/Ceiling fan
4. Chair = Chair/Stool/Seat
5. Child = Boy/Girl/Lad/Doll/Baby
6. Comb = Disentangle, dress
7. Cot = Sofa/Charpai/Bed
8. Almirah = Door/Godrej Shelf/Wooden Case
9. Knife = Cutter/Razer sword/Whittle
10. Book = Register/Volume/Monograph

P. G. I. MEMORY SCALE
Visual Retention Card

P. G. I. MEMORY SCALE
Visual Retention Card

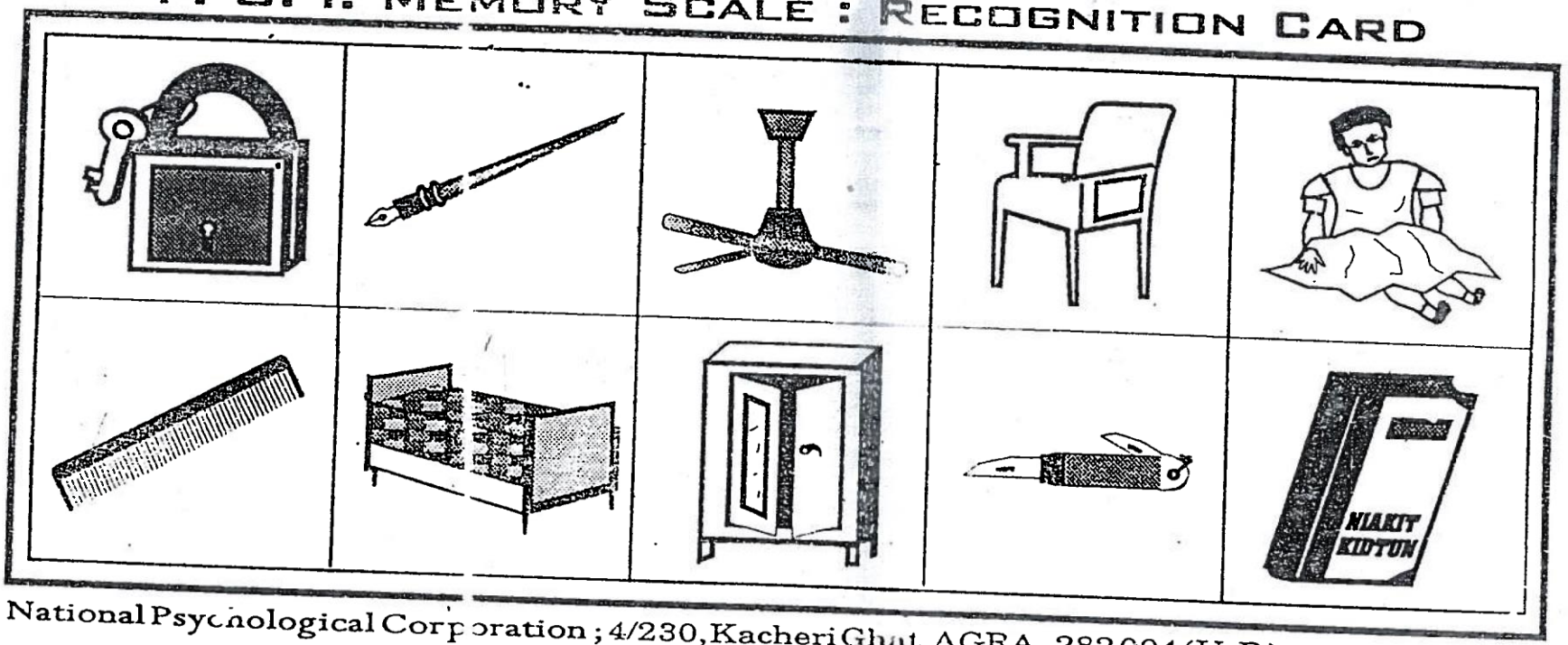
National Psychological Corporation, AGRA - 4

P. G. I. MEMORY SCALE : RECOGNITION CARD



National Psychological Corporation ; 4/230, Kacheri Ghat, AGRA - 282004 (U. P.)

P. G. I. MEMORY SCALE : RECOGNITION CARD



National Psychological Corporation ; 4/230, Kacheri Ghat, AGRA-282 004 (U. P.)

Disease information form

ஆராய்ச்சித் தகவல் தான்

திருநெல்வேலி அரசுப் பொது மருத்துவமனைக்கு வரும் மனநோயாளிகளிடம் மின் அதிர்வு சிகிச்சைக்கு முன்பும் பின்பும் நிகழும் அறியும் ஆற்றல் பற்றிய ஆய்வு இங்கு நடைபெற்று வருகிறது.

மனநோயாளிகளிடம் மின் அதிர்வு சிகிச்சைக்கு முன்பும் பின்பும் நிகழும் அறியும் ஆற்றல் பற்றிய ஆய்வு இந்த ஆராய்ச்சியன் நோக்கம்.

நிங்களும் இநத ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்புகிறோம். இந்த ஆராய்ச்சியில் பங்கேற்பதால் தங்களது.

நோயின் ஆய்வறிக்கையோ அல்லது சிகிச்சையோ பாதிப்புக்கு உள்ளாகாது என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களின் விருப்பத்தின் பேரில் தான் இருக்கிறது. மேலும் நீங்கள் எந்நேரமும் அந்த ஆராய்ச்சியிலிருந்து பின்வாங்கலாம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்தச் சிறப்புப் பரிசோதனையின் முடிவுகள் ஆராய்ச்சியின் போது அல்லது ஆராய்ச்சியின் முடிவின் போது தங்களுக்கு அறிவிக்கப்படும் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

ஆராய்ச்சியாளர் கையொப்பம் பங்கேற்பவர் கையொப்பம்

தேதி

Copy of informed Consent

ஆராய்ச்சி ஒப்புதல் கடிதம்

ஆராய்ச்சித் தலைப்பு : மனநோயாளிகளிடம் மின் அதிர்வு சிகிச்சைக்கு முன்பும் பின்பும் நிகழும் அறியும் ஆற்றல் பற்றிய ஆய்வு.

பெயர்: தேதி :

வயது: உள்நோயாளி எண்:

பால்: ஆராய்ச்சிச் சேர்க்கை எண்:

இந்த ஆராய்ச்சியின் விவரங்களும் அதன் நோக்கமும் எனக்கு முழுமையாகவும் தெளிவாகவும் விளக்கப் பட்டன.

எனக்கு விளக்கப்பட்ட விஷயங்களைப் புரிந்து கொண்டு நான் எனது சம்மதத்தைத் தருகிறேன்.

மனநோயாளிகளிடம் மின் அதிர்வு சிகிச்சைக்கு முன்பும் பின்பும் நிகழும் அறியும் ஆற்றல் பற்றிய ஆய்வு. என்னும் ஆராய்ச்சியில் பங்கேற்க நான் சம்மதம் தெரிவிக்கிறேன்.

இந்த ஆராய்ச்சியில் பிறர் நிர்பந்தமின்றி என் சொந்த விருப்பத்தின் பேரில் நான் பங்கு பெறுகிறேன் மற்றும் நான் இந்த ஆராய்ச்சியிலிருந்துது எந்நேரமும் விலகலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்து கொண்டேன்.

நான் என்னுடைய சுயநினைவுடன் மற்றும் முழு சுதந்திரத்துடன் இந்த மருத்துவ ஆராய்ச்சியில் என்னைச் சேர்த்துக் கொள்ளச் சம்மதிக்கிறேன்.

கையொப்பம்